

**WILL OUR FINAL YEARS BE GOLDEN? MORTALITY BY ALZHEIMER'S  
DISEASE IN THE UNITED STATES**

A Dissertation

by

MARY ANN DAVIS

Submitted to the Office of Graduate Studies of  
Texas A&M University  
in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

May 2006

Major Subject: Sociology

© 2006

MARY ANN DAVIS

ALL RIGHTS RESERVED

**WILL OUR FINAL YEARS BE GOLDEN? MORTALITY BY  
ALZHEIMER'S DISEASE IN THE UNITED STATES**

A Dissertation

by

MARY ANN DAVIS

Submitted to the Office of Graduate Studies of  
Texas A&M University  
in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

Approval by:

Chair of Committee,  
Committee Members,

Dudley L. Poston, Jr.  
Edward Murguia  
Jane Sell  
Cruz Torres  
Holly Foster  
Mark Fossett

Head of Department,

May 2006

Major Subject: Sociology

## **ABSTRACT**

Will Our Final Years Be Golden? Mortality by Alzheimer's Disease in the United States.

(May 2006)

Mary Ann Davis, B.A., Lamar University; M.S.S.W.,

The University of Texas at Arlington

Chair of Advisory Committee: Dr. Dudley L. Poston, Jr.

Alzheimer's disease (AD) is the fifth leading cause of death among the elderly. This study uses National Center for Health Statistics (NCHS) Multiple Cause of Death data for the United States for the years 1998 to 2002, examining the 9.5 million death records of all decedents of age 60 and over, and determines their incidence of AD. Seven independent variables are used: age, sex, race, ethnicity, marital status, education level and whether or not they lived in a metropolitan area. This study uses logistic regression, modeling five nested models, to determine the likelihood of mortality by AD and the direction of the relationship between AD and each of the variables. A Bayesian analysis, used to determine the best fit model, found that the full model was the best fit.

The major findings of the study are that the incidence of AD increases significantly with increasing age in decedents aged 60-90. However, this peaks for decedents aged 85-89. Those who survive past age 90 begin to have a lesser likelihood of mortality by AD. With the exceptions of marital status and education, the hypotheses were supported. Females are more likely to die of AD than males. NonHispanic Whites are more likely to die of AD than Hispanics and NonHispanic Blacks. There is an

increased risk of dying in a nursing home if one dies of AD. Future research as outlined above is needed to learn further about this fifth leading cause of mortality of those over age 60.

## **DEDICATION**

To my husband Ron and my children, Matt and Ellen.

## ACKNOWLEDGEMENTS

First and foremost I would like to acknowledge the guidance and support from Dr. Dudley L. Poston, Jr. From my first class at TAMU I have been directed by his mantra of “demography is destiny”. His encouragement and knowledge of the field directed my research. I appreciated his immediate response to questions. His dedication to his students and his support of their research has been inspirational. I would also like to thank the other committee members. Dr. Jane Sell was always in the lab available for support and advice. Drs. Holly Foster, Cruz Torres, and Edward Murguia provided valuable input into making the final dissertation more complete.

I would also like to thank my children: Matthew and Ellen and my siblings Marilyn, Elizabeth, Frank, Barbara and Claire. Their support and understanding enabled me to concentrate on my research while often ignoring their needs and family events.

Finally, I want to thank my husband, Ron. He has supported me emotionally and financially throughout our marriage but even more so through this doctorate process. I am especially appreciative of his grammatical editing skills, which improved my dissertation.

## TABLE OF CONTENTS

	Page
ABSTRACT.....	iii
DEDICATION.....	iv
ACKNOWLEDGEMENTS.....	vi
TABLE OF CONTENTS.....	vii
LIST OF FIGURES .....	x
LIST OF TABLES.....	xi
 CHAPTER	
I    INTRODUCTION .....	1
Background .....	1
Epidemiological Transition Theory .....	3
Rectangularization of Mortality.....	5
Life Span Is Indefinite .....	6
Alzheimer's Disease.....	9
Population Projections through 2050.....	13
The Extent of Mortality by AD.....	14
Overview of Research.....	17
II   LITERATURE REVIEW.....	19
Theoretical Literature about Life Expectancy and Life Span.....	19
Literature Concerning the Relationship of Sex and AD.....	27
Literature Concerning the Relationship of Education Level and AD.....	31
Literature Concerning the Relationship of Marital Status and AD .....	34
Literature Concerning the Relationship of Metropolitan Status and AD .....	37
Literature Concerning the Relationship of Ethnicity and AD.....	37
Literature Concerning Morbidity and AD .....	45



CHAPTER	Page
Hypotheses Suggested by Literature Review .....	47
III DATA AND METHODOLOGY .....	52
Data .....	52
The Current U. S. Death Certificate.....	56
The International Classification of Diseases.....	57
Variables .....	60
Methodology .....	62
Logistic Regression.....	62
Bayesian Model .....	65
IV RESULTS .....	66
Hypotheses .....	66
Descriptive Analyses .....	67
Logistic Regressions .....	72
Bayesian Measures of Model Fit .....	74
Hypotheses .....	75
Conclusion .....	82
V CONCLUSIONS.....	84
Overview and Summary .....	84
Implications.....	94
Implications for Policy Development for Services ....	97
Provide Care in Less Expensive Manner .....	98
Prevent or Delay the Onset of AD .....	102
Lifestyle Issues.....	103
Future Research .....	104
Conclusion .....	108
REFERENCES .....	111
APPENDIX A.....	123
APPENDIX B .....	129
APPENDIX C .....	131
APPENDIX D.....	133
APPENDIX E .....	134

	Page
APPENDIX F .....	138
APPENDIX G.....	145
VITA.....	187

## LIST OF FIGURES

Figure	Page
A.1    Magnetic Resonance Imaging (MRI) of Brain Atrophy in Alzheimer's Disease.....	125
A.2    Population Pyramid Summary for the United States .....	126
A.3    Percentage of Mortality by Alzheimer's Disease 2001 by Age.....	127
A.4    Cause of Mortality among Decedents Age 60's in the U.S. 2001 .....	127
A.5    Cause of Mortality among Decedents Age 80's in the U.S. 2001 .....	128
G.1    Frequency of Mortality by Age: All Causes, AD and AD as Any Condition, Decedents Over Age 60, United States 1998-2002.....	157
G.2    Frequency of Mortality by Ethnicity in Decedents Over Age 60 in the United States, 1998-2002 .....	157

## LIST OF TABLES

Table	Page
A.1 Midyear Population U. S., 2000, 2025 and 2050 by Age 60+ & Sex.....	123
A.2 Projected Dependency Ratios for U. S. 2000, 2025 and 2050.....	124
A.3 Percentages of Mortality by AD by Age in the U. S. in 2001.....	124
G.1.A Frequency of Decedents Over 60 by Top Causes of Mortality United States 1998-2002.....	146
G.1.B Frequency Distribution of Decedents, Over Age 60, by Age and Ethnicity, United States 1998-2002.....	147
G.1.C Frequency of Decedents Aged 60 and Over by Ethnicity, Mortality by All Causes, AD and AD as Any Condition in the United States 1998-2002 .....	148
G.1.D Frequency of Decedents by Age and Sex, All Causes, AD and AD as Any, in Decedents Over Age 60 in the United States, 1998-2002.....	149
G.1.E Frequency of Decedents Over Age 60 by Marital Status Mortality All Causes and AD, in the United States, 1998-2002.....	150
G.1.F Frequency of Decedents Over Age 60 by Marital Status Mortality All Causes and AD as Any Condition, in the United States, 1998-2002.....	151
G.1.G Frequency of Decedents Over Age 60, by Education Mortality by All Causes, United States, 1998-2002.....	152
G.1.H Frequency of Decedents Over Age 60, by Education, Mortality by AD and AD as Any Condition, United States 1998-2002.....	153

Table	Page
G.1.I Frequency of Decedents Over Age 60, by Metropolitan Status, Mortality by AD, United States, 1998-2002.....	154
G.1.J Frequency of Decedents Over Age 60, by Metropolitan Status, Mortality by AD as Any Condition, United States 1998-2002.....	154
G.1.K Frequency of Decedents Over Age 60 by Place of Death, United States 1998-2002.....	155
G.1.L Frequency of Decedents Over Age 60 by Place of Death, AD and AD as Any Condition, United States 1998-2002.....	156
G.2.A Coefficients and Percent Change in Odds Ratio, AD as the Underlying Condition in Decedents Aged 60-64, United States 1998-2002.....	158
G.2.B Coefficients and Percent Change in Odds Ratio, AD as the Underlying Condition in Decedents Aged 65-69, United States 1998-2002.....	159
G.2.C Coefficients and Percent Change in Odds Ratio, AD as the Underlying Condition in Decedents Aged 70-74, United States 1998-2002.....	160
G.2.D Coefficients and Percent Change in Odds Ratio, AD as the Underlying Condition in Decedents Aged 75-79, United States 1998-2002.....	161
G.2.E Coefficients and Percent Change in Odds Ratio, AD as the Underlying Condition in Decedents Aged 80-84, United States 1998-2002.....	162
G.2.F Coefficients and Percent Change in Odds Ratio, AD as the Underlying Condition in Decedents Aged 85-89, United States 1998-2002.....	163
G.2.G Coefficients and Percent Change in Odds Ratio, AD as the Underlying Condition in Decedents Aged 90-94, United States 1998-2002.....	164

Table	Page
G.2.H Coefficients and Percent Change in Odds Ratio, AD as the Underlying Condition in Decedents Aged 95-99, United States 1998-2002.....	165
G.2.I Coefficients and Percent Change in Odds Ratio, AD as the Underlying Condition in Decedents Aged 100-104, United States 1998-2002.....	166
G. 3.A Coefficients and Percent Change in Odds Ratio, AD as Any Condition in Decedents Aged 60-64, United States 1998-2002.....	167
G. 3.B Coefficients and Percent Change in Odds Ratio, AD as Any Condition in Decedents Aged 65-69, United States, 1998-2002.....	168
G. 3.C Coefficients and Percent Change in Odds Ratio, AD as Any Condition in Decedents Aged 70-74, United States, 1998-2002.....	169
G. 3.D Coefficients and Percent Change in Odds Ratio, AD as Any Condition in Decedents Aged 75-79, United States, 1998-2002.....	170
G. 3.E Coefficients and Percent Change in Odds Ratio, AD as Any Condition in Decedents Aged 80-84, United States, 1998-2002.....	171
G. 3.F Coefficients and Percent Change in Odds Ratio, AD as Any Condition in Decedents Aged 85-89, United States, 1998-2002.....	172
G. 3.G Coefficients and Percent Change in Odds Ratio, AD as Any Condition in Decedents Aged 90-94, United States, 1998-2002.....	173
G. 3.H Coefficients and Percent Change in Odds Ratio, AD as Any Condition in Decedents Aged 95-99, United States, 1998-2002.....	174
G. 3.I Coefficients and Percent Change in Odds Ratio, AD as Any Condition in Decedents Aged 100-104, United State, 1998-2002.....	175

Table	Page
G. 4.A Coefficients and Percent Change in Odds Ratio Dying of AD Compared to Dying of Other Chronic Diseases in Decedents Aged 60-64, United States 1998-2002.....	176
G. 4.B Coefficients and Percent Change in Odds Ratio, Dying of AD Compared to Dying of Other Chronic Diseases in Decedents Aged 65-69, United States 1998-2002.....	177
G.4.C Coefficients and Percent Change in Odds Ratio, Dying of AD Compared to Dying of Other Chronic Diseases in Decedents Aged 70-74, United States 1998-2002.....	178
G.4.D Coefficients and Percent Change in Odds Ratio, Dying of AD Compared to Dying of Other Chronic Diseases in Decedents Aged 75-79, United States 1998-2002.....	179
G.4.E Coefficients and Percent Change in Odds Ratio, Dying of AD Compared to Dying of Other Chronic Diseases in Decedents Aged 80-84, United States 1998-2002.....	180
G.4.F Coefficients and Percent Change in Odds Ratio, Dying of AD Compared to Dying of Other Chronic Diseases in Decedents Aged 85-89, United States 1998-2002.....	181
G.4.G Coefficients and Percent Change in Odds Ratio, Dying of AD Compared to Dying of Other Chronic Diseases in Decedents Aged 90-94, United States 1998-2002.....	182
G.4.H Coefficients and Percent Change in Odds Ratio, Dying of AD Compared to Dying of Other Chronic Diseases in Decedents Aged 95-99, United States 1998-2002.....	183
G.4.I Coefficients and Percent Change in Odds Ratio, Dying of AD Compared to Dying of Other Chronic Diseases in Decedents Aged 100-104, United States 1998-2002.....	184
G.5.A Coefficients and Percent Change in Odds Ratio AD as the Underlying Condition in Decedents Aged 60-105+, United States 1998-2002.....	185
G.5.B Coefficients and Percent Change in Odds Ratio AD as Any Condition in Decedents Aged 60-105+, United States 1998-2002.....	185

Table	Page
G.5.C Coefficients and Percent Change in Odds Ratio AD as Underlying Condition in Decedents Aged 60-105+, Mexican and Cuban Comparisons, United States 1998-2002.....	186
G.5.D Coefficients and Percent Change in Odds Ratio AD as Underlying Condition in Decedents Aged 60-105+, by Ethnicity and Age, United States 1998-2002.....	186



## CHAPTER I

### INTRODUCTION

#### Background

This dissertation explores one of the devastating outcomes for the elderly, the mortality caused by Alzheimer's disease and dementia (AD). Chapter I introduces the research question regarding the extent of mortality, and morbidity, by AD among elderly decedents, over sixty years of age. Three theoretical perspectives of mortality; a rectangularization of mortality, Epidemiological Transition theory and the perspective that life span is indefinite provide the foundation for data analysis of morbidity and mortality in the elderly in the United States. Second, is a description of Alzheimer's disease and dementia, a group of chronic and degenerative diseases increasing in the elderly population. Third, is a description of the population in the United States showing current projections for the aging population through 2050. These projections are compared to those of other countries to show such trends in an aging population are global. Next is the research question exploring the extent of mortality by AD in the over sixty population as well as the rationale for presenting morbidity data to provide a clearer picture of AD. The chapter concludes with a brief outline of the remainder of the dissertation.

The title of this dissertation questions the quality of life among the elderly. The popular picture of the elderly, as depicted by Shakespeare in *As You Like It*, is of a

demented person who is mindless, forgetful and childlike requiring total care, much like the person who is diagnosed with Alzheimer's or dementia:

The last scene of all,  
That ends this strange eventful history,  
Is second childishness and mere oblivion,  
Sans teeth, sans eyes, sans taste, sans everything.  
(<http://www.tech.mit.edu/Shakespeare>).

Senility and dementia are now seen as a disease instead of the natural aging process. A critical issue is how widespread is AD in the elderly population? Dalsania (2004) notes that recent studies have shown that at age sixty, one percent of the population is found to have dementia however by age eighty-five it is estimated that 50percent-80 percent of the population will develop dementia. Since modern man has a longer life span than at any other time in history, we can investigate the mortality and morbidity of the elderly and answer questions of what is normal aging and whether with increased longevity there will be increased disability.

We are facing global issues of an aging population. Later this chapter describes global demographics. Projections of the aging population estimate that by 2050 between 20 to 33 percent of the population will be over age 60. Realistically, although research is making progress in treatment and cure of specific chronic and degenerative diseases that affect the elderly, one cannot expect that life of the elderly will be without morbidity. Accordingly this study has selected one of the chronic and degenerative disease groups, Alzheimer's disease and dementia, to explore the extent of mortality and morbidity.

Alzheimer's disease and dementia (AD) was selected for three reasons. First, AD has become the eighth leading cause of mortality in the elderly, over 65 population in the United States (Hoyert, 1996). Projections of morbidity from AD are even greater with

Dalsania (2004) projecting that by age eighty-five 30 to 50 percent of the population will develop dementia. Brookmeyer and colleagues (1998) estimated that 4.5 million in the U.S. have AD. Even though AD ranks fifth in incidence they noted that AD ranks third in expense due to the length of length of morbidity ranging from 5 to 20 years along with the broad range of services required by those with AD, usually culminating in Nursing home care (Brookmeyer et al.,1998.) Third, is the question of whether there are social demographic issues that influence mortality by AD. Many demographers (Rogers et al.1987, Rogers et al. 1996, Rogers et al. 2000, Rogers et al. 2005 and Schoenborn, 1986) have addressed the effects of social demographic factors such as education, income, ethnicity, social class, medical care access on mortality. This study will address those social demographic variables which influence biological disease processes.

A brief presentation of three theoretical explanations of mortality sets the stage for an understanding of mortality issues: Epidemiological Transition theory, a rectangularization of mortality, and the perspective that life span is indefinite.

### **Epidemiological Transition Theory**

The first theoretical explanation for mortality is Epidemiological Transition theory. This theory was postulated by Omran (1971), consisting of three stages. The first was the Age of Pestilence and Famine the primary causes of mortality were influenza, pneumonia, smallpox, tuberculosis and other related diseases with high infant and childhood mortality and a life expectancy averaging between 20 and 40 years. In developed countries this stage persisted until around 1875. The second stage is the Age of Receding Pandemics. During this stage there is a decline in mortality due to improved sanitation, increases in standards of living and public health, resulting in a steady increase

in life expectancy to between 30 and 50 years. According to Rogers and Hackenberg (1987) the stage of receding pandemics was approximately 1875 to 1930. During the third stage mortality declined due to medical advances in the prevention and treatment of infectious diseases. The remaining causes of mortality are the chronic degenerative diseases, heart disease, cancer and stroke. During this recent stage the life expectancy at birth has risen rapidly so that fertility has been the primary factor in population growth as life expectancy exceeds 70 years (Omran 1971). Olshansky and Ault (1986) report about three fourths of deaths in the advanced years are the result of degenerative diseases. Rogers and Hackenberg (1987) added a fourth 'hybristic stage' where mortality is heavily influenced by individual behavior or life style choices, and deaths are due to social pathologies such as accidents, alcoholism, suicide and homicide as well as life style issues such as smoking and diet. This dissertation later focuses on two social variables, educational level and marital status.

Caldwell (2001) compares the mortality transition of Epidemiological and Demographic Transition theories. The Demographic Transition Theory predominates in explaining population changes in fertility related to modernity. Prior to modernity, there is a high mortality stage which he labels as pretransitional. Second, is the early transitional stage with a decline in mortality due to a combination of hygiene and public health lowering mortality caused by infectious diseases. These declines in mortality began with the youngest, both infants and children, so that in the 185 years between 1780 and 1965 both male and female life expectancies doubled. Third, is the late transitional stage in which mortality is caused primarily by degenerative diseases. In Australia, Canada and Sweden the decline in mortality was attributed to a decline in death from

heart disease; in France and Japan the greatest declines were attributed to a decline in strokes. In the fourth stage, mortality is further reduced because of the delay or reductions in mortality from degenerative causes (Caldwell 2001).

### **Rectangularization of Mortality**

The second theoretical explanation for mortality is the rectangularization of the mortality curve that occurred with modern health practices. As early as 1825 Benjamin Gompertz developed a mathematical formula depicting mortality by age (Rogers et al. 2005), claiming a law of mortality which was depicted as a slope with rates of mortality increasing with age. However, in modern times deaths have been compressed into the oldest old ages. This causes a narrow age range of death appearing not as a gentle slope but as a rectangle. Demographers (Carnes and Olshansky 1993, Olshansky 2003, Olshansky and Carnes 1994, Olshansky et al. 2001, Olshansky et al. 1990, Olshansky et al. 2004) have argued, that we can anticipate a life expectancy of around age 85 or 90 due to senescence, a biological limit to life span. They further argue that even if there are medical advances to increase longevity those who survive due to a cure or treatment of one specific disease will be frail, evidencing morbidity of other disease processes (Olshansky et al. 1990).

Although there have been dramatic increases in life expectancy over the last century these have primarily been changes in mortality risks in infancy, childhood and early adult life. Since these young have already had their life span increased we cannot anticipate such dramatic changes in the future. Unless there is a change in fundamental aging, with increased vulnerability to all common causes of death, life span will not be increased by more than 15 years even if all aging-related causes of death, such as cancer

and heart diseases, are eliminated (Olshansky et al. 2001). Olshansky and colleagues (2004) posit that our search for the magic anti-aging pill leaves us open to “snake oil salesmen” and engaging in risky behavior such as starvation diets. Although starvation diets increase longevity in laboratory studies of other species, they do not readily transfer to humans.

There are no life-style changes, surgical procedures, vitamins, antioxidants, hormones, or techniques of genetic engineering available today with the capacity to repeat the gains in life expectancy that were achieved during the 20<sup>th</sup> century. If there is going to be another quantum leap in life expectancy at birth (20 to 30 years or more), these large gains will have to come from adding decades of life to the lives of people who reach the ages of 70 and older. Modifying endogenous biological processes to achieve this goal, although theoretically possible, will be much harder than reducing children’s death rates from infectious and parasitic diseases. (Olshansky et al. 2001).

Thus, from the perspective of the rectangularization of mortality, one would expect that in the 80’s mortality will continue to increase. Specifically, mortality from AD would continue with the age eighty plus decedents having increasing mortality from AD. Because the theoretical assumption that with senescence there is increased mortality from a combination of causes, an increase would be especially evident if the dependent variable of AD is expanded from the underlying cause of mortality to include all who have AD as one of the twenty conditions of mortality.

### **Life Span Is Indefinite**

The third theoretical perspective that life span is indefinite is a challenge to both Gompertz’s “law of mortality”, which depicts an acceleration of mortality with increasing age, and the rectangularization of mortality. The perspective that life span is indefinite argues that a definite life span is a faulty theoretical concept as it is based on the life span of a single human. The longest known life span is 122 years and five months

authenticated as the age of Frenchwoman Jeanne Calment who died in 1997 (McFalls 2003). This record could be broken by one person who lives to 122 and six months. Thus, since a single human who lives longer than Jeanne Calment can change the life span, we cannot define the life span of humans.

Vaupel (2001) notes that prior to the 19<sup>th</sup> Century only a scattered few individuals survived past 100. There were countries with over one million in population with no documented centenarians (and no supercentenarians, aged 110 and over). However, as we entered this century, there were over 100,000 documented centenarians. Beginning with the first documented supercentenarian, Katherine Plunket, who died at age 111 in 1932 in Northern Ireland; we now have begun to document supercentenarians. With the supercentenarians we question the validity of their age, requiring collaborative documentation prior to accepting claims of age (Vaupel 2001, Vaupel et al. 1998).

Ahlburg and Vaupel (1990) argue that our projections for life expectancy are based on conservative forecasting. They argue that mortality rates have declined at a rate of 1 percent to 2 percent per year in developed countries, especially the mortality rates of the age 65 + population. They assume that if this mortality decrease continues at a 2 percent progression, in 2080 we could expect a life expectancy of 100 years for females and 96 for males (Ahlburg and Vaupel 1990).

If life expectancy was approaching a biological limit one would assume that the mortality rates of the oldest old would tend to be higher in countries with higher rates of the oldest old. However Vaupel (1997) found that countries with the oldest old, France, Japan and Sweden, show a slowing of the mortality rates in the oldest old. Vaupel, Director of the Max Planck Institute for Demographic Research, argues that life

expectancy has been rising at a regular linear pace over the last 160 years. For example whereas Swedish women held the record in 1840 with a life expectancy of 45 years and in 2000 Japanese women held the record for life expectancy at 85 years (Horiuchi and Wilmoth (1998). This increase occurred at a steady rate of almost three months per year. Demographers (Vaupel et al. 1998; Horiuchi and Wilmoth 1998) found that mortality in the elderly goes through three stages; namely a deceleration of mortality after age 80, a mortality plateau between ages 80 to 105 and an actual decline in mortality in the highest ages, over 110. Manton and colleagues (1991) argue that even with the interdependence of diseases as we progress in treating specific diseases such as AD, we are altering senescence.

Since longevity factors are biological issues which should be available for cross species analysis, Vaupel and Carey agreed to collaborate on a large scale life table of the Mediterranean fruit fly. The human aging population is difficult to analyze in a laboratory setting due to ethical issues of experimentation involving subjects in starvation and deprivation environments as well as there being only a small number of supercenturions available for analysis. The Mediterranean fruit fly project, based in Tapachula, Mexico, allowed for a database of over 5.6 million subjects under controlled situations. They were able to expose flies to multiple conditions not possible with human subjects. Using such a large number of subjects Carey found subjects who survived to the oldest old age. The analysis of the indeterminacy of life span research documented, under optimal laboratory conditions, that longevity can be over three times the expected life span of the given species (Carey 2003).



This dissertation will only use five years of data. Thus it is beyond the scope of this project to address the theoretical perspective that life span is indeterminate. However, if life span is indeterminate, mortality from the chronic and degenerative disease of AD should follow the mortality curve of decreasing mortality from AD in the over 80 population. By combining five years of mortality data from the National Center for Health Statistics this study will have a large number of decedents over age 80, 90 and 100 so to be able to analyze the mortality in the oldest old.

### **Alzheimer's Disease**

Mortality from chronic and degenerative diseases includes Alzheimer's disease and dementia as one of the leading causes of mortality in decedents over sixty. Historically, physicians used the terms *senile dementia* and *senility* to describe age related cognitive deterioration. Holstein (2000) notes prior to the mid 70's most physicians considered AD to be rare preferring to use a mixture of diagnoses such as senile dementia, senility, senile psychosis or organic brain syndrome. Part of the reluctance to recognize that AD was a distinct disease is that it was commonly assumed that normal aging was a degenerative process so that loss of memory, decreased cognitive functioning, inattention, and lack of concentration leading to the stage of "second childhood" was but a normal part of aging.

In the past century Alzheimer's disease has been diagnosed as a disease, following its identification by Dr. Alois Alzheimer in a female patient in 1906 (Holstein 2000). As the diagnostic labeling on death certificates has become more standardized with each improvement in diagnostic nomenclature the percentage of the elderly diagnosed with Alzheimer's has increased so that mortality by AD became one of the ten

top causes of death for the over 65 age decedent (Hoyert 1996). AD, a degenerative neurological disease, is classified by the International Classification of Diseases, ICD, 9 and ICD 10 as a mental and behavioral disorder.

Alzheimer's disease is a progressive, degenerative disorder that attacks the brain's nerve cells, or neurons, resulting in loss of memory, thinking and language skills, and behavioral changes. These neurons, which produce the brain chemical, or neurotransmitter, acetylcholine, break connections with other nerve cells and ultimately die. For example, short-term memory fails when Alzheimer's disease first destroys nerve cells in the hippocampus, and language skills and judgment decline when neurons die in the cerebral cortex. Two types of abnormal lesions clog the brains of individuals with Alzheimer's disease: Beta-amyloid plaques—sticky clumps of protein fragments and cellular material that form outside and around neurons; and neurofibrillary tangles—insoluble twisted fibers composed largely of the protein tau that build up inside nerve cells. Although these structures are hallmarks of the disease, scientists are unclear whether they cause it or a byproduct of it. (<http://alzfdn.org/alzheimers/index.shtml>)

AD causes a slow neurological and physiological degeneration. The cognitive degeneration includes memory loss, deterioration in language, comprehension, calculation, learning, and mental functions. The American Psychiatric Association (APA) in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) (1994) defines the clinical syndrome of AD. Although the diagnostic nomenclature of the ICD has changed through editions, the disease Alzheimer's is essentially the same disease as was identified by Dr. Alois Alzheimer in 1906 through an autopsy of a female patient showing the plaques and tangles that today characterize the disease (Holstein, 2000).

The APA DSM-IV notes that AD has two critical aspects; cognitive functioning and the impact of cognitive functioning on daily living activities. First, AD involves cognitive functioning which includes at least two or more of the following domains. The first and most common domain is memory, defined as the ability to learn, retain and

retrieve information. Second is language, both receptive language, encoding information, and expressive language, the ability to communicate verbally or by gestures with others. Third is visual-spatial ability; the ability to judge distance and orientation. Wandering and getting lost or losing items are primary symptoms of an impaired visual spatial ability. Fourth is the executive function that involves planning, organizing sequencing and abstracting. Next is the impact of cognitive functioning on daily living activities such as dressing, eating, toileting, grooming and walking. The cognitively impaired person is no longer able to be independent in daily living activities requiring increasing caretaking as the disease progresses. Finally, there are changes in personality and behavior which may include moodiness, affect lability, aggression, wandering and sleep disruption (APA DSM-IV 1994).

A difficulty in diagnosing AD is due to its insidious, slow progression. With normal aging memory is maintained, with a slight delay in recall of newly learned material (Dalsania 2004). Mild cognitive impairment, with isolated amnesia or memory impairment while maintaining all other domains, may accompany a number of illnesses, and improve as the illness is treated. For example depression may cause mild cognitive impairment which will improve with treatment of the depression with medication. However with AD there is a progression from the cognitive functioning of normal aging, to mild cognitive impairment, to dementia, to death. The progression is variable but seems to be a stepwise progression at a rate of losing 10 to 15 percent of functioning per year. Although there is no cure for AD there are medications that, if taken early may slow the progression (Dalsania 2004).

Initially a diagnosis of AD was only confirmed after death by autopsy of the brain, as an autopsied brain revealed the characteristic neurological tangles and plaque and significant atrophy of AD. Hoyert (1996) notes that research in the 1970's indicated that 60 to 70 percent of the suspected cases were confirmed through autopsy and that in the 1990's more than 90 percent of suspected cases were confirmed by autopsy. Current diagnostic techniques, including neurological testing and scans allow diagnosis in the living. For example a magnetic resonance imaging (MRI) clearly reveals AD without autopsy. Figure A.1 in the Appendix is a MRI which shows the atrophy and loss of brain mass in the progression of the disease (<http://www.mayoclinic.com>).

AD is the most frequent cause of dementia. Dementia is also associated with cerebrovascular disease, the second most common cause of dementia; moreover one-third of stroke patients develop dementia. Other causes of dementia include normal pressure hydrocephalus, affecting about 10 percent of adults over age 60; alcohol related dementia/ Korsakoff's syndrome; Parkinson's disease, dementia with Lewy bodies and frontal temporal dementia. Infections may also cause dementias such as AIDS, viral encephalitis, spirochetal disease, Lyme disease, chronic bacterial meningitis and Creutzfeldt-Jacob's disease (CJD) (APA-DSM-IV 1994, Dalsania 2004, Hoyert 1996,). Other dementias will be included in this study because they are also progressive neurological dementias with a similar impact.

One of the critical issues about AD is its increasing effect in an elderly population. Dalsania (2004) notes that recent studies have shown that at age sixty, one percent of the population is found to have dementia, however, by age eighty-five it is estimated that 50 percent to 80 percent of the population will develop dementia. Modern

man has a longer life span than any age making possible a demographic analysis of mortality and morbidity of a late onset disease such as Alzheimer's. This raises the question of how many elderly will be diagnosed with Alzheimer's and dementia at death as well as what will be the morbidity of the elderly. Also addressed is the theoretical question of whether, as demographers (Manton and Gu 2001 and Uhlenberg 1992) have posited, with increased longevity there will be increased disability.

### **Population Projections through 2050**

According to the US Census Bureau, International Population Reports, in 2004, life expectancy at birth in the World was 65 for males and 69 for females. In more developed countries it was 72 and 80, and in less developed countries, 63 and 67. The highest life expectations at birth were in Japan, 78 for males and 85 for females; the lowest were in Sierra Leone, 34 for males, 36 for females. In 2002 globally there were 440 million persons over age 65 which is 7 percent of the total population. The over 65 population is projected to double by 2020 and almost triple by 2050 with 17 percent of the world's population projected to be over age 65. This segment is projected to grow faster than any other age segment due to increased longevity and decreased fertility (US Census Bureau 2004). The United Nations (UN) projections are based on the population that is over 60 and show even more dramatic increases in the elderly. The UN projects that by 2050 there will be nearly two billion over age sixty, or 22 percent of the population. The UN reports global aging trends of median age increasing from 23.5 years in 1950, to 26.4 in 1999 with a projection of 37.8 in 2050. Further the UN projects that nearly one person in three will be over age 60 in 2050 (<http://www.un.org/esa/population/publications/sixbillion/sixbilpart1>).

In 2001 life expectancy in the U.S. was 77 years, ranking 20<sup>th</sup> in the world. In the U. S. during the last century, life expectancy at birth has increased from 47 in the 1900's to 67 in 2000 (McFalls: 2003). This increase coupled with lower fertility is creating an aging population. Descriptive data in Appendix A, Table A.1 from the U.S. Bureau of the Census International Data Base, (IDB) indicate that 3.3 percent of the US population was 80+ in 2000, 4.5 percent are projected to be 80+ in 2025 and 8 percent are projected to be 80+ in 2050.. The IDB Population Pyramid summary for the U.S. in Appendix A, Figure A.2 depicts the current population and trends with the aging of the baby boomers, lower fertility rates and the aging of the population. The numbers for the IDB Pyramids over age 60 are included in Appendix A, Table A.1. Note the projection in 2050 is that the median age is 39.1 and approximately 10 percent of the females are over age 80.

Appendix A, Table A.2 illustrates the U.S. Bureau of the Census IDB projected dependency ratios for the U. S. in 2000 to be 18.75 percent over 65, with projections for 2025 of 29.33 percent over 65 and 2050 with 34.57 percent over 65.

As mentioned earlier Ahlburg and Vaupel (1990) argue that our projections for life expectancy are based on conservative forecasting. They argue that the U. S. Census bureau's projection of a life expectancy of 91 for females and 85 for males in 2080 is low. Since mortality rates have declined at a rate of 1 percent to 2 percent per year in developed if one assumes this progress continues through 2080 one could expect a life expectancy of 100 years for females and 96 for males (Ahlburg and Vaupel 1990).

### **The Extent of Mortality by AD**

This dissertation will address one aspect of increasing disability in the elderly population, specifically mortality and the related morbidity caused by AD. Hoyert (1996)

posited that AD as a cause of mortality has increased significantly from 1979 to 1991. “Between 1979 and 1991 the age-adjusted death rate for Alzheimer’s disease increased twelvefold from 2.5 to 29.3 per 100,000 population age 65 and over” (Hoyert 1996). This pattern is probably due to better diagnosis of AD and better medical certification than to an actual increase in disease (Hoyert 1996). AD has become the eighth leading cause of mortality in the over 65 population.

It must be noted that there is a suspected underreporting of AD as a cause of mortality on death certificates, data which will be used in this dissertation. Burns and colleagues (1990) report that in 30 percent of the cases studied there was no mention of dementia on the death certificate recommending that a higher autopsy rate would increase the accuracy of reporting of AD on death certificates. Hoyert (1996) argues that neither AD nor other dementias are mentioned on a quarter to one third of the death certificates of diagnosed persons. Hoyert notes that AD is underreported on death certificates for a number of reasons: first AD may be present but not diagnosed; second, AD may be diagnosed but not be considered to have caused the death; third the person with AD may only be recognized when the disease is in its final stages; fourth, the individual with AD in the final stages is susceptible to other chronic conditions and infectious conditions which are more easily reported by medical personnel as conditions contributing to death (Hoyert 1996).

Hebert, Scherr, Bienias, Bennett and Evans (2003) studied the incidence of AD using three adjacent neighborhoods in Chicago. Seventy-nine percent of the residents participated. AD incidence was measured among 3838 persons. Of these, 835 met the criteria established by the National Institute of Neurological and Communicative

Disorders and Stroke. This study estimated 4.5 million in the U. S. with AD; 0.3 million or 7 percent were between ages 65 and 74; 2.4 million or 53 percent were between ages 75 and 84; and 1.8 million or 40 percent were 85 years of age or older. They estimated that in 2010 there will be 5.1 million persons with AD and by 2050 there will be between 1.3 million and 16.0 millions with AD due to the rapid growth in these oldest age groups (Hebert, et. al, 2003:1119)

Although mortality data does have limitations as were mentioned earlier, with an underreporting of mortality by AD recognized by Hoyert (1996), the data are the documented causes of mortality. Refer to Appendix A, Figure A.3 for an initial analysis of the National Center for Health Statistics (NCHS) Multiple cause of Death files in 2001(Davis et al. 2004). Refer to Appendix A, Figure A.3 for the percentages of mortality by AD by age over 60 years. The incidence of AD increases from .71 percent for male decedents and .04 percent for females decedents in their 60's to 2.3 percent for male decedents and 2.78 percent for female decedents in the 70's. Decedents in the 80's continued to have increased mortality from AD; the 4.48 percent of male and 5.52 percent of female decedents in their 80's died of AD. The growth in mortality by AD slows in the 90's and 100's to 4.61 percent for males and 5.86 percent for females in the 90's and 3.21 percent for the males and 4.07 percent for females in the 100's. This increase in AD as a cause of mortality is clarified by Figures 4 and 5 which depict the top causes of mortality in decedents two age groups, age 60's and age 80's respectively. Appendix A, Figure A.4 shows that at for decedents aged 60's AD is the twelfth leading cause of mortality. Appendix A, Figure A.5 shows that in decedents aged 80's the percentage has increased so that AD is the fifth leading cause of mortality.



Uhlenberg (1992) notes that unless there is significant progress in research, the increased numbers of the oldest-old will lead to increased numbers of elderly with disabilities requiring long-term care. The implications of mortality from late onset degenerative diseases must be considered in conjunction with the age structure of the population as decreases in fertility rates, and increases in life expectancy during the last century have profound implications for the population. Therefore, this topic is a particularly timely one in that the large cohort of “baby boomers” is reaching the age at risk for dementia. This creates the need for a demographic analysis to prepare for the significant financial impact of morbidity by AD in this cohort.

There is a gap in research pertaining to the incidence of AD in the oldest old, those over 90. This dissertation will use mortality data to address this gap by combining five years of data to have sufficient numbers of those who die after age 90 and after age 100, addressing whether the increase in AD that begins in the 60’s continues in the oldest old or whether there is a critical period of AD incidence.

### **Overview of Research**

Following this introductory chapter, Chapter II reviews the relevant literature on mortality by Alzheimer’s Disease and describe current demographic research regarding mortality issues related to chronic and degenerative diseases affecting the elderly. Chapter III will discuss the data and methods of analysis. The data are Multiple Cause of Mortality data for the years 1998 to 2002 from the National Center for Health Statistics, NCHS. Descriptive analysis is used first to study variables affecting the aged decedents including the main causes of mortality by age. Descriptive analysis also depicts mortality differences by sex, ethnicity, marital status, education level, metropolitan status

and place of death. Next the logistic regression depicts the odds of dying of Alzheimer's disease versus other causes of mortality using the independent variables. Chapter IV provides the results of the analysis of the NCHS death certificate data. Chapter V summarizes the main findings of the dissertation, presents implications for policy development and suggestions for future research.

## **CHAPTER II**

### **LITERATURE REVIEW**

This chapter reviews relevant literature on mortality by Alzheimer's disease, describing current demographic research regarding mortality issues related to chronic and degenerative diseases affecting the elderly. The AD literature is organized into categories related to the variables to be addressed in this dissertation: theoretical literature about life expectancy and life span; literature regarding the demographic variables of age, sex, marital status, race and ethnicity, education level; and general morbidity. A bibliographic review was undertaken using such internet web searches of JSTOR, Science Direct, Popline, Cambridge Scientific Abstracts, and Sociological Collection. A brief synthesis of the literature for each variable will follow, which will be used in chapter three to support the research hypotheses.

#### **Theoretical Literature about Life Expectancy and Life Span**

Epidemiological Transition theory, espoused by Omran (1971) posits three main stages: the Age of Pestilence and Famine, the Age of Receding Pandemics and the Age of Chronic and Degenerative Diseases. According to Rogers and Hackenberg (1987) the United States is in a fourth 'hybristic stage' with mortality heavily influenced by individual behavior or life style choices. In this stage most deaths are due to social pathologies such as accidents, alcoholism, suicide and homicide, as well as life style issues such as smoking and diet.

The bulk of literature pertaining to the incidence of AD appears to substantiate Epidemiological Transition Theory, with increases in AD related to the increases in numbers in the aging population. The difficulty in obtaining valid statistics globally not

withstanding, Wilmo and colleagues (2003), used worldwide United Nations data, based on the medium fertility variant, to project the population of the world. They calculated the number of cases with dementia as multiplications of the age and class-specific dementia: “1% in age group 60-64 years, 1.5% in 65-69 years, 3% in 70-74 years, 6% in 75-79 years, 13% in 80-84 years 34% in 90-94% and 45% in  $\geq 95$  years (Wilmo 2003, 64).” Because of global aging, they estimated the number of demented people will increase from 25.5 million in 2000, to 63 million in 2030, and to 114 million by 2050. Although there were some international conflicting reports of AD incidence (e.g. China and Nigeria reporting a lower portion of dementia than in Europe and North America), other studies show global prevalence similar to the Western rates with AD increasing as the population ages (Wilmo 2003).

The trend of increasing AD has been supported by research. The Center for Disease Control (CDC) estimated that in the United States the incidence of AD doubles every 5 years after age 65 with approximately 10 percent of adults  $\geq 65$  years, and 47 percent of adults  $\geq 85$  diagnosed with AD (CDC: 2003, 104). As the population aged and the techniques for the diagnosis of AD were refined, the death rate from AD increased from 0.4 per 100,000 in 1979 to 4.2 per 100,000 in 1987 (CDC: 1990,1). Sahyoun and colleagues (2001), examining death certificates beginning in 1933, noted an increase in mortality by AD as the population aged. AD was ranked among the top 10 leading causes of death in 1994. Hoyert (1996) also found an increase from 857 AD deaths in 1979 to 14,112 AD deaths in 1991. She explained the increase in AD caused mortality as being due to multiple factors including an increase in the population at risk for AD,

improvement in the diagnosis of AD, and an increase in medical personnel reporting AD and other dementias on death certificates.

Other researchers (Antuono and Beyer 1999, Dalsania 2004, Gambassi and colleagues 1999, Giovanni and colleagues 1999, Hoyert 1996, Nocera and colleagues 2003, Pollen 2000) have shown support for the positive association between AD and age. Dalsania's percentages were lower than those of the CDC. He found that at age 60 one percent of the population has dementia. However, by age 85 an estimated 30 to 50 percent of the population has dementia (Dalsania 2004). Gambassi and colleagues (1999) agree that about 50 percent at age 85+ have AD. Giovanni and colleagues (1999) suggest a prevalence of AD in about 50 percent of the over 85 population in the US, estimating nearly 4 million people to have a diagnosis of AD, which would make it the fourth leading cause of death in the elderly.

Nocera and colleagues (2003) have shown that the rate of AD increases with age with an occurrence of 9 to 10 percent at age 65+ and 30-50 percent at age 85+. The incidence rates used to describe newly diagnosed cases were 0.5 percent at age 65 and 8 percent at age 85. Pollen (2000) extended the incidence of AD to the lower age of the 50's, by noting that the risk of AD doubles every five years between ages 50 to 80. Hoyert (1996) noted that National Center for Health Statistics mortality data from 1979-1991 showed that rates for the 85+ age group were 19.3 times those for the 65-75 age groups.

Other studies have focused on projections from specific neighborhoods. Hebert and colleagues (2003) studied the incidence of AD in three neighborhoods in Chicago, with a seventy-nine percent participation rate. Of the 3,838 persons in the study, 835 met

the criteria for AD established by the National Institute of Neurological and Communicative Disorders and Stroke. From this study, they estimated that of the 4.5 million persons in the U. S. diagnosed with AD, 0.3 million or 7 percent were between ages 65 and 74; 2.4 million or 53 percent were between ages 75 and 84; and 1.8 million or 40 percent were 85 years of age or older. They estimated that in 2010 there will be 5.1 million persons with AD and by 2050 there will be between 1.3 million and 16.0 millions due to the rapid growth in these oldest age groups (Hebert, et. al, 2003:1119)

Comorbidity of AD with other chronic and degenerative diseases also effects mortality. With the aging of the world's population, 80 percent of those aged 65 and over will have one chronic condition, and 50 percent will have at least two chronic conditions (CDC 2003). Hoyert (1996) argued that the comorbidity of AD with other chronic conditions has lead to the underreporting of AD on death certificates. Even if AD was diagnosed and may have contributed to death, the physicians were accustomed to reporting one cause of death, the overt reason for the death, rather than the contributing chronic conditions.

Ganguli and colleagues (2005) compared death certificate conditions with diagnostics to confirm suspected underreporting of AD on the death certificates. They found only 12.3 percent of those diagnosed with AD had it reported as a condition of death. The extensive comorbidity might be a cause of this underreporting. They also found that cancer deaths were underreported in those with AD which might reflect either that AD or cancer were competing risks or that there is a lower detection of cancer in those with AD (Ganguli et al. 2005).

Mortality in the hybristic stage of the Epidemiological Transition theory tends to be heavily influenced by individual behavior or life style choices (Rogers and Hackenberg 1987). This hybristic stage is supported in the Cache County Study (Miech and colleagues 2002 and Tschanz and colleagues 2004). Cache County, Utah was selected for study because it has an elderly population with the highest life expectancy in the United States. Over 90% of the population belongs to the Church of Jesus Christ of Latter-day Saints, a church which prohibits such life style choices known to increase risk factors for heart disease and cancer, as smoking and drinking alcoholic beverages. These beliefs also endorse life style choices, such as stable marital unions and close knit families, which also support longevity.

Tschanz and colleagues (2004) monitored deaths in Cache County, Utah, using vital statistic records and newspaper obituaries of 355 individuals with dementia and 4,328 with no dementia. They were screened using psychological, medical and neurological exams, and an informant interview. As was hypothesized they had lower incidences of AD than would be estimated by national age projections suggesting that life style indeed influences AD (Miech and colleagues 2002 and Tschanz and colleagues 2004).

The research of Antuono and Beyer (1999) also supports the hybristic stage; Their data suggested that only 5 percent of AD may be genetically determined while the remaining 95 percent may be attributed to personal choice risk factors such as education, nutrition and incidence of vascular disease which is itself alterable by healthy life choices.

The second theoretical perspective is the rectangularization of mortality. Some demographers (Carnes and Olshansky 1993, Olshansky 2003, Olshansky and Carnes 1994, Olshansky et al. 2001, Olshansky et al. 1990, Olshansky et al. 2004) have posited the existence of senescence, that is a biological limit to life span, namely a life expectancy of around age 85 or 90. They argue medical advances to increase longevity will leave frail survivors susceptible to morbidity of other disease processes (Olshansky et al. 1990).

Wilmoth and Horiuchi (1999) have described the rectangularization of mortality as a compression of mortality at older ages so that the survival begins to appear more rectangular suggesting there is a distinct upper limit to human life expectancy. Even though life expectancy has increased from the 30's in historical times, to the upper 70's in modern times, patterns of mortality reduction appear to be slowing. Wilmoth and Horiuchi (1999) agree with Epidemiological theory that initially the greatest decreases in mortality were aged infancy to five years. Once a population reaches age 70 and above mortality decline tends to slow.

Research by Brookmeyer and colleagues (2002) and Ganguli and colleagues (2005), would seem to support the rectangularization of mortality theory. Ganguli and colleagues (2005), in a fifteen year epidemiological study which included 1000 person-years, found that the age of onset and the duration of survival differed by age. Elderly persons, over age 85, who were diagnosed with AD, survived for fewer years than those who were diagnosed prior to age 75.

Brookmeyer and colleagues (2002) also found that length of survival after diagnosis depends on the age at onset, or original diagnosis, which would support a



rectangularization theory. If AD is diagnosed in the 60's or 70's the average length of survival is 7 to 10 years. However, if diagnosed in the 90's, the average length of survival is 3 years or less. Brookmeyer and colleagues (2002) have noted that if prevention strategies or treatments could allay symptoms even for five years, the current goal of research, there could be significant reduction in morbidity by AD.

Another theoretical perspective is that the life span is indefinite. Demographers (Vaupel et al. 1998; Horiuchi and Wilmoth 1998) supporting this perspective have posited that mortality in the elderly goes through three stages; a deceleration of mortality after age 80, a mortality plateau between ages 80-105, and an actual decline in mortality in the highest ages, over 110. This theory does not refute Epidemiological Transition theory; it merely suggests that the life span in the modern societies is indeterminate.

Several research findings support the theory that the life span of the oldest old is increasing, pointing to the fact that those who survive the risks of mortality at earlier ages have an expanded life (Gatz et al. 2000). Miech and colleagues (2002) support the theory that life span is indefinite in that the pattern of mortality by AD, along with other chronic and degenerative diseases, declines in extreme old age. For both sexes the incidence of AD was lowest in the oldest old. They studied 185 individuals with dementia among 3,308 participants. They found that the incidence of AD increased until age 85 to 90 but appeared to decline after age 93 for men and age 97 for women. Miech and colleagues (2002) concluded that the incidence of AD increased with age but peaked and declined among the extremely old, those over 100.

Brookmeyer and colleagues (1998) studied the incidence of AD using age-specific incidence rates from epidemiological studies in Boston, Framingham, Rochester

and Baltimore. They agree that the incidence of AD grows exponentially with a doubling time of 4.9 years. However, they question whether this growth continues to the oldest old where rates appear to plateau and then decline. They noted that there are limited data regarding the incidence of AD beyond age 95 other than estimates based on extrapolations from current models. Data from Brookmeyer and colleagues (2002) also support this theory, suggesting a decline of AD in the oldest old.

Gambassi and colleagues (1999) support the argument that AD does not have a linear relationship with age, agreeing that there is a decrease in AD in the oldest old. They found that autopsies of those who die of AD at advanced ages show fewer senile plaques and neurofibrillary tangles, the key autopsy diagnostics for AD.

Gatz and colleagues (2000) found that rates of AD tend to increase exponentially with age with approximately 0.5 cases per 100 being in age group 60-65, which increases to 123 cases per 100 in the age group 80-85. They speculated a plateau after age 90-94 years of age (Gatz et al. 2000).

Perhaps this argument has been most clearly stated by Ritchie and Kildea (1995) who argue that although the onset of AD is age related, in that it occurs within a specific age range, it is not caused by the aging process. Due to limited numbers of the  $\geq 80$ , they did a meta analysis of data from 12 studies. They found that the rate of increase in AD begins in the 60's and continues to a point when it begins to decline in age range 80-84 and then levels off at around age 95. They noted that, to date, single studies have not included sufficient numbers of the  $\geq 80$  population to address this population (Ritchie and Kildea 1995). Other researchers (Gao et al. 1998, Hofman et al. 1991) also attempted to address the gap of research in the oldest old through meta analyses however the numbers

of those over 95 were limited. These studies would indicate a gap in the literature on the incidence of AD in the oldest old.

### **Literature Concerning the Relationship of Sex and AD**

Incidence data of AD by sex include sex differences in both morbidity and mortality. All agree that AD occurs in both sexes and is age related. Thus the question is whether the incidence of AD is higher in females owing to more females surviving to older ages. Demographers (Christensen and colleagues 2001 and Carey 2003) have addressed the greater survival of females across species due to multiple causations, including the advantage of the X chromosome. Briefly, males have two chromosomes, X and Y, and females have two X chromosomes. Males must have both X and Y chromosomes. However, if one of the female X chromosomes is deficient the other X chromosome can be used, maximizing the possibility of having healthier genetics. Among mammals the life span record holder is most often a female.

Bonsignore and colleagues (2002) noted that epidemiological surveys on the sex differential have been inconsistent although the trend was for higher mortality by AD in females. This higher incidence was partially explained by three factors. First, females have greater longevity and were thus more likely to contract an age related disease. Second, females have lower education than age-matched male, and education level was a risk associated with AD. Third, depression was also indicated as an AD risk factor and females had a higher incidence of this psychological disorder. They sampled 1,628 patients including those affected with AD or depression and matched control subjects. Fifty-one percent were females and had a mean education level of 9.3 years. They found that males and females showed the same risk of AD and suggested that in order to study

the sexual incidence of AD subjects should be matched in number, age and educational level (Bonsignore et al. 2002).

Heun and Kockler (2002) studied 146 patients with AD, 168 patients with major depression, 136 age –matched subjects from the general population and 718 first degree relatives. They found most of the cognitive impairment of AD was from preexisting gender differences in specific cognitive impairment. For example there were gender differences in visual spatial tests with women typically scoring lower, and women had lower education levels than males. Although women had better verbal skills than men, they did not find differences in the language decline by sex.

Larson and colleagues (2004) in an observational study from 1987-1996, studied AD following initial diagnosis in a base population of 23,000 persons aged 60+ in Seattle, Washington, of whom 521 were diagnosed with AD. This sample was obtained from the clients of a health maintenance organization. They were persons who had access to health services which may have biased the sample by excluding those with lower socioeconomic conditions. They found that men had poorer survival in all age groups surviving only 4.2 years following diagnosis, whereas women survived 5.7 years. The sex gap did vary by age with a median life expectancy of 4.4 additional years for a 70 year man with AD compared to 8.0 years for a woman. However, overall, men with AD had half the life expectancy of women with AD.

Ganguli and colleagues (2005), in a fifteen year epidemiological study which included 1,000 person-years, found that the age of onset and the duration of survival differed by sex. The mean age of AD for males was 80.1 years with duration of 6.3 years. In women the mean age of onset of AD was 80.3 with duration until death of 5.8 years.

Hoyert (1996) found that although initially it appears that more females than males die of AD, the age adjusted death rates for males were greater than those for females. Hoyert (1996) noted that female rates increased almost every year from 1979 to 1991 while for men increases appear to have stopped around 1987 (Hoyert, 1996). Katzman and Bick (2000) also noted that although initially AD was seen as a female disease, their research did not find this pattern.

Dodge and colleagues (2003) studied a population sample of 1,201 individuals, in the Monongahela Valley of Pennsylvania. At age 70 and every 2 years thereafter they compared those with AD to those without. They found that a diagnosis of AD shortened life expectancy in both males and females. Although women with AD lived longer than men with AD they spent more years than males with increased disability. Both sexes also had an average of 6 to 7 more activities of daily living (ADL) impairments than those who were nondemented. Although women had a greater comorbidity, as well as a more rapid progression of AD, they still lived longer than men (Dodge et al. 2003).

There is a popular view that hormone replacement therapy (HRT) protects post menopausal women from AD. However, this has not been confirmed by epidemiological research. Helmer and colleagues (2000) studied 3,675 participants in a French cohort, aged 65+ from 1988-1998. Two hundred and eighty-one were actively diagnosed with AD, with a mean age of onset of 82.3 years. Although the onset of dementia was difficult to pinpoint, their results showed that women with AD had longer survival than men. The effect of sex decreased with age, with greater differences in sex at age 75 than at age 85 and above. Helmer and colleagues (2000) found that few women in their study had taken HRT so this could not explain the longer survival of women with AD.

Zandi and colleagues (2002) also using the Cache County Study tested whether the use of hormone replacement therapy (HRT) would decrease the risk of AD. They had small numbers of HRT users in the study so their findings were inconclusive. However HRT only appeared to be beneficial if taken over 10 years (Zandi et al.2002).

Antuono and Beyer (1999) agree that the epidemiological studies on HRT were inconclusive. HRT studies to date have not addressed the coexistence of other predisposing factors such as education, access to health care, and lower cerebrovascular health risk factors.

Miech and colleagues (2002) in “The Cache County Study” found that the incidence of AD tended to decline in the early 90’s for men and in the later 90’s for women. The incidence of AD in women was much higher than in men after age 85. Tschanz and colleagues (2004) in “The Cache County Study” found male sex and older age were associated with a shorter length of survival following diagnosis with AD; however the interactions of sex with dementia and other risk factors were not significant.

Barnes and colleagues (2005) studied the clinical manifestations of AD in males and females using a population of 1000 Catholic nuns, priests and brother in a longitudinal Religious Orders study, obtaining annual clinical evaluations and autopsies at death. Of the original 1,000 subjects, 196 had died and 181 had undergone brain autopsy. They found that if they controlled for age and education level, sex was not related to the odds of AD. However, they did find stronger clinical manifestations of AD in women. Barnes and colleagues speculated that females might lack some protective factor, such as estrogen receptors, which might increase their vulnerability to AD (Barnes et al. 2005).

Lapane and colleagues (2001) studied 2,838 males and 6,385 females  $\leq 65$  who were newly admitted to nursing homes in five U. S. States with a diagnosis of probable AD. They found that men were more likely to die of AD, though women with AD showed more severe impairment and an accelerated rate of decline (Lapane et al. 2001). They found that women with AD have a better prognosis than men, when age, race and severity of dementia were controlled.

### **Literature Concerning the Relationship of Education Level and AD**

Most studies support the assertion that higher education levels were either protective against AD or delay the onset of symptoms. Those studies that do not find educational level to be significant were those with a homogenous population where education level does not vary.

Mortality research in general noted that there is a tendency for those with higher education to have increased health and longer life span. Many demographers (Crimmins and Saito 2001, Rogers, Hummer, Nam and Peters 1996 and Stern et al. 1994) have noted that mortality in general was higher among those with low levels of education, low incomes and unemployment. Since education level was attained at a young age there were lifelong effects from educational status. Those with higher education were more likely to have better access to a healthier lifestyle and to live longer. They found that persons with lower education have shorter lives, and that racial differences in total life expectancy were greatest with lowest levels of education. Black males with low education have a 19.9 year lower life expectancy than high educated whites (Crimmins 2001:1634) When one looks at mortality by social class, Katzman and Bick (2000) have shown that education appears to protect one from AD.

Antuono and Beyer (1999) found that low educational achievement in early life is a risk factor related to the development of AD in later life. Of the 600 nuns in the United States Nun Study, the 14 nuns who developed AD had low education levels. Their autobiographies, written prior to the onset of AD, showed low linguistic ability and poor cognitive functioning. Antuono and Beyer questioned whether higher education may identify those with greater cognitive reserves who will be less likely to develop AD. Another speculation is that those with higher education may have greater longevity with less disability from the AD.

Bennett (2004) presented a preliminary report of a longitudinal Religious Orders Study of 950 nuns, brothers, and priests. The participants underwent detailed annual tests of a variety of memory and cognitive skills and at death also donated their brains for autopsy. Initial autopsy of 130 decedents showed that although both groups had the same number of plaques, those with higher education showed relatively less mental decline. He postulated that mentally challenging activities often provide a reserve that protects against mental decline.

Freeman and Martin (1999), using the 1983 and 1993 panels of the Survey of Income and Program Participation, found that improvements in education and adaptive functioning of the aged tend to increase together. The pathways from increased education begin in early life with increases in income, as well as fewer environmental stressors and lower risk factors due to access to health care and preventative health care. Persons who were not high school graduates had twice the risk of functional limitations in later life. They postulated that increases in education level may continue, however, possibly not as dramatically as in recent decades (Freeman and Martin 1999).



Scarmeas and colleagues (2001) and Stern and Colleagues (1994) were associated with the Taub Institute for Research in Alzheimer's disease and the Aging Brain. They studied 1,772 individuals age 65+, followed longitudinally for 7 years. They found those with higher education appeared to have a reserve protecting against AD. Since leisure activities were related to education and occupation, they expanded education to include leisure recreation and social activities. They found that those with high leisure activities whether they were physical, social or intellectual, had the lowest relative risk of AD, suggesting that increasing leisure activities might reduce the risk of AD.

Heininger (2000) agreed that most studies suggest that lack of education or a lifelong history of lower cognitive activity is related indirectly to the incidence of AD. The "use it or lose it" advantage of the higher educated posited that having a lifelong pattern of cognitive activity built a brain reserve that protected against AD. There also may be a diagnostic bias in that psychological tests favor those with higher education. He suggested that there may be a bias in the person with low education may have low cognitive functioning so that AD can be detected sooner. However, his overall findings were that the effect of education was low and that education level was not significantly associated with survival.

Tschanz and colleagues (2004) in "The Cache County Study" found that years of education was not significant in mortality risk. However the population studied was a homogeneous, white and of similar education and religion. Over 82 percent of the participants in the Cache county study had  $\geq 12$  years of education.

Gatz (2000) studied 129 pairs of Swedish twins in which one twin in each pair had dementia. He compared these twins with 249 other twins with dementia and 498

healthy twins. Gatz found low education positively related to AD. Of the twins with AD, 90.4 percent fit the criteria for low education. However, education was not significant when compared with their healthy twin, suggesting that genetics, not education, had the protective affect.

Wolfson, and colleagues (2001) studied factors of survival following the onset of dementia. They found that education level was not associated with length of survival. However, the Canadian population was homogenous and only had two categories of education, below 8 years and above 8 years.

### **Literature Concerning the Relationship of Marital Status and AD**

Demographic studies support a negative relationship between marital status and AD. However, most studies do not include the oldest old. Fratiglioni and colleagues (2004 ) have postulated three hypotheses to explain the negative relationship between marital status and AD. The first is the cognitive reserve hypothesis. This would posit that marriage selects those who have greater capital such as more education and better health; therefore the married would be less likely to develop AD. Second, is the vascular hypothesis. Dementia is related to cognitive decline following vascular incidents. The married have better health, better diets and were more likely to have a healthier lifestyle and thus be less likely to suffer from vascular dementia. Third, is the stress hypothesis. Those who were married have a support system which minimizes stress as well as offering more coping alternatives. They found that those who were married were more likely to have an active and socially integrated lifestyle in later life which protects against dementia and AD.

Many demographers (Goldman 2001 Rogers 2000) agreed that the underlying processes involved in marriage lead to the healthiest individuals getting married, and that these processes continue so that age graded effects of marriage continue with age. The married usually were physically healthier, more attractive and even taller (taller persons were more likely to be upwardly mobile) than those who were single and never married. Married people also have higher quality occupations, working in less dangerous jobs for fewer hours, with more health insurance benefits. Those who were married have higher education levels. Moreover these factors occur at a younger age so the married have lifelong advantages over those who were single and never married. Married people were less likely to smoke, use drugs, drink, and were less likely to be mentally ill. In old age when there is the increased likelihood of chronic diseases, those who have the social support of marriage were more likely to receive care in their own homes, and if hospitalized more likely to return to their own homes following treatment. The difference in life expectancy appears to be growing so that in Japan, one of the countries with the highest life expectancy, married people have a 15 year life-expectancy advantage over the single (Goldman 2001).

Antuono and Beyer (1999) have also noticed that nutrition is related to AD. The married were more likely to have a healthy diet, which is related to the onset of AD from cardiovascular disease.

Fratiglioni and colleagues (2000) in the Swedish Kingsholmen Project studied 1,203 people who lived in their own homes in Sweden, 176 patients who were diagnosed with AD. They found compared with married people, single people and those living alone had a greater risk of developing AD. They found that satisfaction in contacts with

children, friends, and relatives was more important than the frequency of contact.

However, being single carried the highest risk, with no difference between widowed or divorced people living alone and widowed or divorced people living with others.

Bassuk and Glass (1999) followed 2,812 elderly <65 for 12 years and found that those with increased social ties, including presence of a spouse, monthly contact with at least three relatives or close friends and yearly contact with at least 10 relatives or close friends were less likely to experience cognitive decline. Persons who had no social ties were twice as likely to experience cognitive decline as persons who had 5 or 6 social ties. Seeman and Crimmins (2001) have noted the relationship between social ties and social engagement with dementia and cognitive impairment. Their results showed that higher levels of social support, such as the emotional support of marriage were more strongly related to improved functioning in the early stages of cognitive decline than in the later stages.

Berkman (2000) has noted that even cross species studies show the benefits of family living in maintaining cognitive functioning. Goldman (2001) has shown that monkeys and baboons with higher social advantages, associated with marriage in humans, were less likely to have cardiovascular disease and atherosclerosis and have a better immune system.

Other studies involving marital status were qualitative in nature involving the spouse as caretaker and the relationship of nursing home admission to disability (Corcoran, 2004; Dobalian et al., 2003; Elman and Uhlenberg, 1995; Meuser, 2004; Rogers, et al., 2000; and Uhlenberg, 1992). Elderly females who live alone present the

greatest health risks; however these studies do not address marital status as a specific risk factor in AD.

### **Literature Concerning the Relationship of Metropolitan Status and AD**

There were few studies exploring metropolitan and urban versus rural incidence of AD. Dobalian and Radcliff (2003) analyzed the 1996 Nursing Home Component of AHRQ's Medical Expenditure Panel Survey. They compared the diagnoses of 5,899 residents residing in 815 rural and urban nursing home homes and found no difference in a diagnosis of AD between rural and urban homes.

Other studies pertained to services access. There was less accessibility to services and more limited medical, social and caretaker services available in rural areas.

Geronimus and colleagues (2001) found that rural dwellers lived longer than urban dwellers and the more affluent urban dwellers lived longer than those in high poverty. Poverty, race and environment had multiple interactions on mortality.

Financial considerations also varied with persons in rural areas of the United States more apt to seek expensive nursing home care as the only available service option thereby making AD in rural areas more expensive. Another aspect of rural urban differences was that if persons with AD in rural areas wander, they faced greater environmental dangers than those who wander in an urban setting where their aberrant behavior is more likely to be spotted.

### **Literature Concerning the Relationship of Ethnicity and AD**

The literature about the relationship between race and ethnicity and AD is inconclusive. First, as was mentioned in Chapter I, race and ethnicity involve multiple

risk factors including but not limited to socio-economic level, early life exposure to health care services, education level, as well as exposure to environmental hazards.

Second, racial demographics were a factor in the increased diagnosis of AD which was aging related. According to the Kochanek and colleagues (2004) at the Center for Disease Control (CDC) the black/white age disparity has been decreasing since 1989. They project that the racial differences in AD will change as the number of blacks begins to reach the age at risk, over age 65. Although in 2002 the age adjusted death rate for mortality by AD was 0.7 black to white and 0.5 Hispanic to Non Hispanic white, there was a 5.8% increase in mortality by AD from 2001 to 2002, and 2.4% of mortality was caused by AD in 2002. The comorbidity of both blacks and Hispanics with hypertension, strokes and diabetes, which contributes to vascular dementia, places them at an increased risk of AD as the population ages (Kochanek et al. 2004).

Demographers (Crimmins and Saito 2001, Geronimus et al. 2001, Rogers et al. 1999 and Rogers et al. 2000) have also noted that differences in mortality by race may actually be due to differences in socio-economic level, education level, marital status, hazardous employment and access to health insurance. Geronimus and colleagues found that even economically advantaged blacks have greater mortality than whites of the same economic level. This was likely related to the high incidence of stress related diseases, including heart disease, high blood pressure and strokes among middle-class blacks compared to whites. Myers and colleagues (1989) argued that these stress related diseases were responses to racial oppression.

Ethnicity poses similar ambiguities in that ethnic groups often show more within group than between group differences with the majority population. Some demographers

(Berkman, et. al. 1989, Geronimus et al. 2001 and Manton and Gu (2001) have concluded that the differences in black and white mortality related to health status were largely the result of socio-economic variables. Although disability from chronic and degenerative diseases declined from 1982 to 1999, chronic disability in blacks did not follow this same straight line decrease. Chronic disability increased in black Americans from 1982-1989 though it began to decline from 1989-1994 with a larger decline from 1994-1999. This decrease appeared to follow the same pattern as the increase in education level (Manton and Gu 2001).

A third factor was the black/white mortality crossover which has been researched for the past twenty years. Many researchers (Berkman et al. 1989, Coale and Kisker 1986, Johnson 2000, Lynch and colleagues 2003, Nam 1995 and Preston 1996) have addressed the mortality crossover. Although this research has been questioned, due to possible age under reporting, there was a consistent pattern of mortality crossover in the oldest old. Lynch (2003) found that the age of crossover has increased from age 79 years in 1970 to 87 years in 1992. Coale and Kisker (1986) credit the crossover to the early mortality of the frail so that only robust persons survive.

Research about the impact of race and ethnicity were at times contradictory. First were the studies which have showed no difference in mortality by AD between blacks and whites. Green and colleagues (2001) compared the risk of an AD diagnosis among relatives of whites and African Americans who have a genetic tag of an AD. They followed for 10 years, between 1991 and 2001, 17,639 first-degree biological relatives and 2,474 spouses of 2,339 whites diagnosed with AD probands, and 2,281 first-degree biological relatives and 257 spouses of 255 African American diagnosed with AD

probands. They found that if one controlled for sex, first degree relatives of whites and African Americans, with the genetic tag of AD probands, had the same risk of AD (Green et al. 2002).

Gambassi and colleagues (1999) found that in spite of increased socioeconomic risk factors, minority groups had a reduced risk of AD mortality compared to non Hispanic whites. Gambassi and colleagues conducted a longitudinal study of all Medicare/Medicaid residents in nursing homes in five U.S. states. This population included 9,264 patients age 65+ with AD. They found that other racial or ethnic groups, including African-Americans, had a lower risk of mortality than whites, independent of the severity of the dementia (Gambassi et al. 1999, 62). They also found that the age at onset of AD was difficult to obtain because nearly 80 percent of AD diagnoses were made by lay evaluators. Although confirmed by hospital discharge record documentation, there were inconsistencies.

Other studies showed a lower rate of AD in blacks compared to whites. The CDC has noted that from 1979-1987, although the AD mortality rates for blacks and whites both increased by age, the rates for whites was higher than for blacks in all age ranges (CDC: 1990,1). The CDC has also reported that in California in 1985-1987 of those diagnosed with AD 77.2 percent were white, 9.1 percent were black, and 2.1 percent were Hispanic (CDC: 1990).

Gambassi and colleagues (1999) used a SAGE database of all nursing home residents from 1992-1995 in five U. S. states. This study included 9,264 patients aged 65+ followed for a median of 23 months, with about 70 percent female and 91 percent white. They found that blacks had a reduced risk of mortality compared to whites, even



after adjustment for age, sex and level of impairment. The factors most associated with increased risk of mortality were male sex, age and white race (Gambassi et al. 1999).

There was also the question of whether the black incidence of AD was environmental or genetic. Hendrie and associates (2001) compared American and African black populations in the Indianapolis-Ibadan Dementia Project, a longitudinal study with a baseline survey in 1992-1993 along with follow-up waves, in 1994-1995 and in 1997-1998. The African residents sampled were 2,459 Yoruba residents of Ibadan, Nigeria, without dementia. The African American residents initially sampled were 2,147 residents of Indianapolis, Indiana, without dementia (all aged 65 years or older). They found that the age-standardized rates of AD were significantly lower among the Yoruba, indicating that factors other than race were influencing AD rates (Hendrie et al. 2001).

Heininger (2000) also addresses the issue of genetic predisposition or environmental causes of racial differences in AD by comparing the low incidence of AD among East Africans with African Americans. Although these were racially similar populations, the different environments revealed different levels of AD. He suggests that the Western lifestyle of limited physical exercise and high cholesterol diet set the stage for AD. He noted also that the genetic factors cannot be overlooked. There was a higher incidence of AD in European or American immigrants than in African or Asian immigrants (Heininger 2000).

Saenz and Morales (2005) have suggested that ethnicity has more pronounced effects than race in its comparison with the dominant group as ethnic groups have racial and cultural distinctions as well as economic disadvantages. Although they acknowledge that both race and ethnicity were socially constructed, they make distinctions between

three Latino groups: Mexican Americans, Puerto Ricans and Cubans. The issue was further confused by a changing nomenclature, for example persons of Mexican origin have used ethnic identities of “Mexican”, Mexican American”, Chicano”, “Latino”, and “Hispanic.” Ethnic groups can also alter their ethnicity by self reporting as white. The current trend among statistical agencies was for self identification of race and ethnicity and to allow multiracial identification.

Within ethnic groups demographic patterns differ. The Cubans migrated to the United States in two waves, one in the 1950’s, a higher educated class in a forced permanent exile from Fidel Castro’s communist revolution, and the second the boat people, lower class and criminal immigrants exiled by Castro in the 1980’s as a drain on the Cuban economy. This first group is now elderly. Cubans are an older population, and as an older population a greater risk for aging related chronic and disabling diseases. However, Cubans have higher education and socio-economic status. Mexicans and Puerto Ricans have the youngest populations (Saenz and Morales 2005).

Saenz and Morales (2005) and Rogers and colleagues (2005) have noted that mortality within ethnic groups is affected by several variables including immigration effects such as the self selection of the healthy to migrate. Hummer and associates (1999) noted that the Mexican immigrant has a better diet, closer kinship support and is less likely to smoke and drink than the native born.

Some demographers (Palloni and Morenoff 2001, Abriaído-Lanza et al. 1999, Franzini et al. 2001) have posited the “Salmon effect” of the sick/elderly immigrants returning to native lands leading to the underreporting of mortality. It should be noted that Puerto Ricans can be used to test the Salmon effect because unlike Mexicans, who

will be uncoded if they return to Mexico, Puerto Ricans have U. S. death certificates because Puerto Rico is a U.S. Territory.

Others (Elo and Preston 1992 and Saenz and Rubio 2004) have discussed whether the noted ethnic differences were due to environmental and socioeconomic variables versus genetics. Elo and Preston 1992 have argued that immigrants from less developed countries might experience different mortality rates due to diseases or early adult experiences in underdeveloped countries which predispose them to chronic diseases in later years. Saenz and Rubio (2004) have distinguished between Mexican Hispanics who entered the United States at an early age and the native born population. They found that the foreign born who immigrated after age 65 have the lowest disability rates. Compared to whites and Asians, those who immigrated at age 0-14, with a longer exposure to Western diet and lifestyle risk factors, had the highest disability rates.

The CDC (2004), using 2001 Mortality Data pertaining to different leading causes of death, found that Hispanics compared to Non Hispanic Whites have different leading causes of mortality. For non-Hispanic whites AD was the eighth leading cause of mortality with 49,030 or 2.5 percent of the population dying with AD as the underlying cause of mortality in 2001. For Hispanics AD was not ranked in the top 10 causes of mortality. The CDC noted that in 2001 Hispanics were more likely to die before age 75, which would make them less likely to die of age graded AD (CDC: 2004).

Conversely, the website of the Alzheimer's Association argues that Hispanics were at a greater risk of AD. It predicts that the number of Hispanics with AD will increase by 600% between 2000 and 2050. It is noted that the life expectancy of Hispanics is expected to continue to increase. Since age is associated with AD risk, as

the Hispanic population ages so will the increase of Hispanics with AD. It predicts that by 2050, 16 percent of the total U. S. elderly population will be Hispanic. Hispanics also have higher percentages of other risk factors, related to the comorbidity of AD with other chronic diseases, especially vascular diseases and diabetes. Finally, lower education levels place Hispanics at a greater risk of AD. Ten percent of Hispanic elders have no formal education and 50 percent have  $\leq 8$  years of education ([www.alz.org/Media/newsreleases/2004](http://www.alz.org/Media/newsreleases/2004)).

Saenz and Morales (2005) have also described six Asian groups, namely, Asian Indians, Chinese, Filipinos, Japanese, Koreans and Vietnamese. Due to migratory patterns the oldest Asian groups were the Japanese, with a predominance of females in the aged 65 and above age group, due to the war brides marriages following World War II. Due to low fertility one fifth of the Japanese population was elderly.

There were also cross-cultural issues in the recognition of AD. Poston and Kim (1999) have noted that the incidence in North and South Korea and the United States were similar. Watari and Gatz (2004) compared Korean Americans with other ethnic groups in California and found that compared to European Americans they have underutilized services for AD patients. They hypothesized that the Korean cultural beliefs in Confucianism assign the responsibility for the care of the parents to the eldest son. These beliefs limit help seeking as it would be seen as a stigma. Also a greater respect for the elderly as well as more tolerance for cognitive decline might result in the person with AD remaining undiagnosed in a home care situation for a longer period.

### **Literature Concerning Morbidity and AD**

The literature on morbidity, the disease process of AD, was more qualitative. This section will briefly review literature concerning morbidity from AD as it relates to the variable on the death certificate as Place of death: private residence, nursing home, hospice and home health which are outpatient care and hospital care. First, Mitchell and colleagues (2004) have noted that in nursing home settings those patients with AD have increased mortality, compared to other diagnoses. Covinsky and Yaffe (2004) agree. They followed 521 patients of a Health Maintenance Organization and found that those with AD had half the life expectancy anticipated from life tables.

Wolfson and colleagues (2001), using data from the Canadian Study of Health and Aging, found that the median survival from onset of symptoms was 3.3 years, with a range from 1 to 16+ years. They also found that the younger the age of onset the longer the survival.

Freeman (1993) used the National Nursing Home survey to model the hazard of leaving a nursing home based on covariants including diagnosis of AD and kin. A problem with the study was that she did not know about discharge disposition, whether they left the nursing home to return to family living or death. She found that daughters and wives were more likely to provide care than sons and husbands. Women who have children or spouses have an average three months shorter stay in nursing homes. She found that women with AD have the shortest stay of all persons with terminal conditions, possibly because AD was not diagnosed with certainty until the individual was in an advanced stage.

Rapp and colleagues (2005) studied the neurological functioning of 288 participants with a mean age of 84.5 years; 192 gender and age matched community residents compared to 96 residents from the Jewish Home and Hospital, Bronx, NY. They found that functional disability was greater in nursing home residents than in community dwellers. They also found, controlling for gender, education level and age, that decline was more pronounced in nursing home residents than in community dwellers (Rapp et al. 2005).

Blieszner and Alley (1990) noted that results of the national Long-Term Care Survey found that although almost 85 percent of the 25.5 million elders in 1982 lived independently, about 2 million required assistance with activities of daily living (ADL), with the need for assistance increasing with age. Of the caregivers, 29 percent were daughters, 23 percent were wives, and 19 percent were husbands. Those elderly surveyed preferred to live independently or in their own home. The types of services available include health care services funded by Medicare and Medicaid; institutional care; the older Americans Ace funds; Home repair; Senior Centers; Friendly visitation; Telephone assistance; Home Delivered Meals; Homemaker/Chore services; and Adult Day Care. These programs have financial eligibility limitations so if adults have family members who can provide these services they were often excluded from services via the Older Americans Act. They argued that most families caring for a family member with dementia need respite, day care and support groups and if this were the case, then more demented family members could remain in the community.

Blieszner and Shifflett (1990) studied the relationship of married and parent-child pairs among caretakers of AD. They found that the relationship altered as the symptoms

of the disease increased with the person with AD going through stages from memory loss and confusion to total helplessness to death. They found three stages in the relationships: an initial stage of grief and regret; a second stage of redefinition of expectations; and a third closure stage of accepting that even though the person was physically present he or she was emotionally absent.

Harwood and colleagues (2000) studied the appraisal and psychological well being related to caregiving in 114 family caregivers of AD patients in Miami, Florida. They studied an ethnically mixed population of 56 percent non Hispanic White caregivers and 44 percent Hispanic caregivers. They found that ethnicity was not related to caregiver burden. However there were ethnic differences among the caretakers in that there was a higher percentage of adult daughter caregivers in the Hispanic families compared to spouses in the non-Hispanic white population.

### **Hypotheses Suggested by Literature Review**

This chapter has reviewed literature related to AD as an age graded increasing cause of mortality in the elderly, and the literature related to demographic variables hypothesized to be risk factors for the development of AD or protective of AD. The following are the main hypotheses to be tested in this dissertation. They are drawn primarily from the above literature review; they state the predicted relationships and the directions of the relationships.

This dissertation will compare male and female decedents over age 60 with mortality of Alzheimer's disease versus the other primary causes of mortality in the United States. Because of the issues presented in the literature review by CDC (2003) and Hoyert (1996) of possible under reporting of AD, three versions of AD will be used as

the dependent variable. The first version uses those listed with the underlying cause of mortality as AD; this version assumes that the NCHS criteria were followed by those who complete the death certificate. The second version is introduced because of the issue of comorbidity (CDC 2003, Hoyert (1996) Dalsania 2004, Ganguli et al 2005). This version focuses on those with AD as any of the 20 conditions of mortality listed on the death certificates compared to all decedents over 60 by age and sex. Finally, the third version responds to the issue of senescence, a biological limit to life span (noted in Chapter I), posited by demographers (Carnes and Olshansky 1993, Olshansky 2003, Olshansky and Carnes 1994, Olshansky et al. 2001, Olshansky et al. 1990, Olshansky et al. 2004). This version uses those with AD scored as a dummy variable, compared to all decedents over 60 with a chronic and degenerative disease.

Initially this study planned to use a fourth version of the dependent variable, those who were diagnosed with AD by autopsy. However Hoyert (1996) noted the improvements in diagnostics that occurred between the 1970's through the 1990's so that more than 90 percent of suspected cases were confirmed by autopsy. The Mayo clinic reports that current diagnostic techniques, including magnetic resonance imaging (MRI) clearly reveals AD without autopsy. Since autopsy diagnosis is no longer necessary, this version of AD will not be used (<http://www.mayoclinic.com>).

Eight independent variables will be used: age, sex, race, ethnicity, marital status, education level, living in a metropolitan area and place of death. Age is measured in five-year groups beginning with decedents age 60-64, 65-69, 70-74, 75-79, 80-84, 85-89, 90-94, 95-99, 100-104, 105-109, 110-114, 115-119, 120+. Sex is coded as male or female. Racial categories compiled from the death certificates were Black, White or Asian.



Ethnic categories provided by NCHS were Non-Hispanic White, Non-Hispanic Black and Hispanic. Marital status categories were obtained from NCHS as married, single-never married, widowed and divorced. One limitation of this data is that marital status is reported by informants with not verification of actual status at death or how long the decedent was married, widowed or divorced. Education levels used were elementary school, middle school, high school and college. Place of death categories from the death certificate are outpatient, residence, dead on arrival (doa), inpatient and nursing home.

The following series of hypotheses have been drawn from the literature review presented earlier.

1. Elderly decedents will experience age graded mortality by Alzheimer's disease with a increased risk of AD up to age 90.
2. Elderly decedents who survive past age 90 were less likely to die of AD than decedents in their 70's and 80's. The deceleration of mortality in the oldest old addresses the theoretical viewpoint of longevity, the theory that life span is indefinite. The AD literature, suggests that there is a deceleration of AD mortality at the oldest ages; however it notes that there were limited data concerning the incidence of AD over age 95 other than estimates. One advantage in this dissertation is that it will combine five years of death certificate data from approximately 10,000,000 death certificates with about 80,000-100,000 decedents over age 100. With these data this study can address this gap in the literature. If accepted the results would question the alternative theory of demographers who support the rectangularization of life, with mortality due to senescence at around age 90 or 90.

3. Elderly female decedents will be more likely to die of AD than elderly male decedents. The literature questions the relationship of AD mortality by sex . The consensus is that females live longer so were more likely to be in the age graded risk period of AD, and that the greatest sex differences in AD were under age 85. Researchers suggest that to study the relationship between sex and AD one must control for both age and education level.
4. Elderly decedents, under age 85, with higher education, high school and above will be less likely to die of AD than those with elementary education. Mortality researchers noted that mortality in general is higher among those with low levels of education. The Nun Priest Studies found that low educational achievement in early life is a risk factor related to the development of AD in later life although it is not know if this is due to greater cognitive or a lifetime of increased economic and familial support leading to greater longevity with less disability from the AD.
5. Elderly decedents will experience the mortality crossover effect, that is, blacks who survive to the 80's will achieve greater longevity than whites, applies to AD in that those blacks who survive past their 80's present lower odds of dying of AD than whites.
6. Hispanic populations:
  - a. There is a higher incidence of AD in stable non migrant populations of Hispanics using the Cuban population compared to other Hispanics.
  - b. There is a lower incidence of AD in migratory Hispanic populations where the "Salmon effect" may be evident, that is by comparing those in the Southwestern states to other Hispanics and to NonHispanics.

c. There were differences in mortality by AD among Hispanic populations: Puerto Ricans, Cubans, and Southwestern residents, those within 100 miles of the Mexican Border.

7. Persons who die of AD will more likely be Nursing home residents. The bulk of literature supports that AD is more expensive due to the extensive care required during a long morbidity, ranging from 5 to 20 years. Nursing home as place of death will be used as a way to address this issue. Although there is the limitation that nursing home as place of death does not address the length of stay in the nursing home prior to death.

This chapter has reviewed the literature related to AD as an age graded increasing cause of mortality in the elderly, along with a review of literature related to demographic variables which were seen as risk factors for the development of AD or protective of AD. This literature review was followed by the hypotheses to be tested in this dissertation. The next chapter, Chapter III, will present the data and methodology.

## CHAPTER III

### DATA AND METHODOLOGY

#### Data

This dissertation investigates dementia mortality among the elderly decedents (persons aged 60 and over) in the United States. The data are death certificates of those decedents over age 60, who died in the United States between 1998 and 2002. The primary advantage of using these data is the large numbers allow for a study of the oldest old.

#### *Death Certificate Data Source*

Death certificate data are available from the National Center from Health Statistics (NCHS) in Hyattsville, Maryland. The NCHS provides health information from vital statistics, birth and death certificates, as well as national survey data. This material is available to the public and health researchers. The data used in this dissertation are the NCHS Multiple Cause of Death Files (from 1998-2002), a compilation of the death certificates filed for all those who died in the U. S. in this five year period. There were 12,009,528 death certificates in this period and 9,643,607, for decedents dying after age 60. In 1998 there were 2,340,708 records; in 1999 there were 2,394,871; in 2000 there were 2,407,193; in 2001 there were 2,419,960 and in 2002 there were 2,446,796. It should be noted that no period effects are noted in this five year period. Although there were approximately 2,600 deaths due to 9/11 most of these decedents were age 30 to 35 and thus did not appreciably affect mortality or the mortality

of the over age 60 decedents in this study. This compilation of five years of data permits the analysis of the oldest old, a gap noted in the literature review in Chapter II.

Hetzel (1997) provided a historical context for the current NCHS mortality data. Beginning in 1790 the United States required a census every ten years to assure equal representation in the legislature. However, the need for vital statistics was not realized by the founding fathers. In the founding period there were neither universal registration laws nor permanent legal records covering an entire state. Vital statistics were initially under local control and used to document paternity and lineage for the protection of individual property rights.

On the continent during this period there was a base of support for vital statistic data, especially death data, in order to develop a better understanding of diseases and mortality. John Graunt (1620-1674), an Englishman, began this movement with his analysis of the *Bills of Mortality*. He created a basic life table which promoted a better understanding of mortality caused by the plagues in London (Poston, et al 2005).

Hetzel (1997) noted that Americans, following English trends, also began to study vital records to explore the relationships between diseases and mortality. In the early settlements diseases, such as malaria, dysentery, smallpox and typhoid fever, could spread rapidly killing a local population. In 1721 Cotton Mather used mortality statistics to address the smallpox epidemic in Boston. He was able to show the increased survival of those inoculated against smallpox by presenting data indicating that one in sixty inoculated died of smallpox, whereas one in six of those who were not inoculated died. Subsequent epidemics led to individual U. S. cities introducing health departments. In 1797 Massachusetts passed the first state law creating a local health department.

On the continent advances in European vital statistics developed in response to the Industrial revolution. Louis Villermé (1782-1872) showed that crowding and unsanitary conditions in Parisian neighborhoods were related to the spread of disease. There was a widespread belief that progress in public health services would be the primary weapon against pandemics such as the cholera epidemic that by the 1830's had traveled a route following trade and immigrants from Asia through Russia, Germany, Britain, Canada and the United States. Shylock and Siegel (1973) noted that panic, caused by fear of pandemics, along with humanitarianism, helped both statisticians and the authorities realize the need for precise universal vital statistics. From the onset vital statistics were part of the scientific movement positing that an empirical approach could improve living conditions. In 1836 Great Britain passed a central registration law and the United States followed with a similar registration act in 1842 and 1844. These acts required the central filing of death certificates by states on standardized forms with specific causes of mortality (Hetzel 1997).

In 1846 the American Medical Association created a committee to improve the registration of births, marriages and deaths. Initially there was disagreement about how to use mortality data. One group emphasized contagion, diseases causing death entering the country through seaports and spread by animals or people. They believed that quarantine would prevent contagion. This group promoted data for quarantine and were instrumental in the passing of a Congressional act in 1878 for the registration of mortality by contagious diseases, labeled as "notifiable diseases;" such as cholera, smallpox, plague, and yellow fever. In 1879 a clause was added to the act requiring the collection

of data about the “notifiable diseases” from port areas to further track contagion from foreign ports.

The second group was composed of promoters of public health. Medical officers in the civil war noted that disease killed more people than did weapons, with troop movements spreading diseases such as typhoid. They posited that diseases were caused by sewage, garbage and polluted water as the result of overcrowding and poverty. The American Public Health Association, founded in 1872, used vital statistics to promote public health reforms, showing the decrease in mortality by contagious diseases through sanitation reforms. Specific notifiable disease reports began in a few cities and states prior to 1900, and by 1925 all states reported them regularly. The public health movement was advanced by medical discoveries: Koch isolated the cause of cholera, Gaffky discovered the cause of typhoid fever, Smith and Kilborne explored the control of malaria and yellow fever, and German and French bacteriologists discovered the cause of diphtheria. Pasteur invented the process for pasteurization of milk. All of these discoveries vastly decreased infant mortality and changed the acceptance of the need for mortality data (Hetzel 1997).

In 1880 the first death certificates were standardized so that enumeration could be mechanized, easing the accuracy of the count by the use of the Hollerith mechanical calculator, adapted from the textile industry for use in mortality (Hetzel 1997). The mechanization of the 1890 census led to standardized death certificates. In the 1900 census the census office, designated as Bureau of the Census in 1903, requested that each reporting area use a standardized death certificate, and around 1913 the Bureau of the Census appointed representatives in local health departments to direct the standardization

of death certificates. The increased mortality during WWI led to increased efforts at standardization; however death certificate standardization occurred on a state by state basis. The final state to join both birth and mortality registration was Texas in 1933. During the 1930's, mortality registration shifted to the Public Health Departments; the emphasis was on the control of contagious diseases as public sanitation was nationally accepted. The onset of WWII led to a need for the reliable registration of births and deaths for the defense industry. In 1946 the National Office of Vital Statistics was established as a part of the Public Health Service. In 1942 the gathering and reporting of mortality statistics were assumed by the Division of Public Health Methods and in 1949 by the National Office of Vital Statistics, where they remain today within the National Center for Health Statistics (NCHS) (Hetzel 1997).

### **The Current U. S. Death Certificate**

Appendix B shows the current death certificate, with completion instructions. By law the funeral director compiles all data except for the cause of death, which is completed by the attending physician. In the case of unnatural death the coroner completes the cause, even if there is an attending physician. Current death certificates have two categories for reporting cause of death: immediate cause and underlying cause. Physicians and coroners receive training to increase the accuracy of distinguishing between an immediate cause of death, i.e. the final disease, injury or complication, versus the underlying cause of death, i.e. the disease, injury or complication that started the chain leading to death. The death certificate allows the reporting of up to twenty conditions so that all other significant diseases, conditions, or injuries that contributed to death are listed.



Appendix C lists the Data File Description from 1999-2002 and from 1989-1998. This shows the items that are coded from the death certificates which are available for analysis through the NCHS Multiple cause of death files. The data reported in 1998, via the Data File Description 1989-98 have slight variations, notably, that 1994 was the last year that reported whether or not an autopsy was performed as part of the death certificate file. Hoyert (2001) reported a decline in autopsies since the Joint Commission on the Accreditation of Hospitals eliminated autopsy requirements for hospital certification in 1971. In 1961, 41 percent of hospital deaths had autopsies; this decreased to between 5 and 10 percent by the middle of the 1990's. Autopsies are usually performed only when there is an undetermined or unnatural cause of death (Hoyert 2001). Therefore this variable will not be used here as a diagnostic criterion. Data to be used in this study include geographic and metropolitan status information, age, education, race and ethnicity, marital status, sex and underlying and multiple causes of death.

### **The International Classification of Diseases**

The diagnostic naming of cause of mortality requires an explanation. Health statisticians recognized that a systematic diagnostic nomenclature was required for comparing data across times and geographic entities. In 1893 Jacques Bertillon prepared the first international classification of diseases, the *Bertillon Classification or International List of Causes of Death* which was accepted in the United States in 1898. The International Classification of diseases resulted from the Bertillon Classification and is considered the “gold standard” of disease nomenclature, used universally. The ICD has been revised approximately every ten years with advances in medical science. Revisions included: ICD-1, 1900-1909; ICD-2, 1910-20; ICD-3, 1921-29; ICD-4,

1930-38; ICD-5, 1939-48; ICD-6, 1949-57; ICD-7, 1958-67; ICDA-8, 1968-78; ICD-9, 1979-1998 and ICD-10, 1999-current (NCHS 1998).

This dissertation uses two versions of the ICD which demonstrate the refinements in diagnosis from 1979 when ICD-9 was introduced until 1994 when ICD-10 was issued. ICD 10 was accepted for use in the U. S. in 1999; however ICD-9 continues to be used in morbidity surveys. The ICD-9 is also used in the 1998 NCHS mortality files. The ICD-10 is used in the NCHS mortality files from 1999-2002.

The ICD-9 is a two volume set with 4,000 disease categories whereas the ICD-10 is a three volume set with 8,000 categories. The changes between the two were primarily a refinement of categories. The ICD hierarchical positions remain the same so that the diagnostic categories used in this dissertation will not be affected by the changes between ICD-9 and ICD-10. There are coding differences with the ICD-9 using numerical coding and the ICD-10 using an alpha numeric code (<http://www.openclinical.org/medTermICD>).

Appendix D lists the chapter titles in ICD-9 and ICD-10 and describes the nomenclature for each chapter. The chronic and degenerative diseases studied do not have significant nomenclature changes in the major categories of disease; most of the refinement is with respect to distinctions within disease category. For example in ICD-9, V is Mental Disorders (290–319) which is reclassified in ICD-10 as Mental/behavioral disorders (F00–F99). AD is coded under VI Diseases nervous/sense organs (320–389) in ICD-9 as 331 and in ICD-10 in the Diseases of nervous system (G00–G99) as G20.

Appendix E lists the 113 Cause of Mortality Recodes from the NCHS. Since ICD-9 has 4,000 categories and ICD-10 has 8,000 categories the CDC recommends using the

113 Cause of Death recode used in The International Comparison of data. Appendix F is a compilation of the top causes of mortality for both sexes and all races for all ages in five year age groups from age 60-100+. Although there is minor variation in the top causes of mortality this dissertation will use the top 15 causes of mortality:

1. Diseases of heart (I00-I09,I11,I13,I20-I51)
2. Cerebrovascular diseases (I60-I69); Atherosclerosis (I70)
3. Malignant neoplasms (C00-C97)
4. Influenza and pneumonia (J10-J18)
5. Alzheimer's disease (G30) and Parkinson's disease (G20-G21)
6. Chronic lower respiratory diseases (J40-J47) and Pneumonitis due to solids and liquids (J69)
7. Accidents (unintentional injuries) (V01-X59,Y85-Y86)
8. Nephritis, nephrotic syndrome and nephrosis (N00-N07,N17-N19,N25-N27)
9. Diabetes mellitus (E10-E14)
10. Essential (primary) hypertension and hypertensive renal disease (I10,I12)
11. Septicemia (A40-A41)
12. Intentional self-harm (suicide) (\*U03,X60-X84,Y87.0);
13. All other causes (Residual). (Although the cause Assault or homicide,

\*U01-\*U02,X85-Y09,Y87.1, is one of the top causes among all ages, it is not used here because it is not a top cause among the elderly population.)

## **Variables**

This dissertation addresses the possibility of undercounting AD with three treatments of AD as the dependent variable. The first treatment is AD as the underlying cause of mortality. In the logistic regression AD is treated as a dummy variable, that is either a decedent dies of AD or other causes. This compares AD to all other causes of mortality of decedents over 60 categorized by age and sex. The second treatment is also a dummy variable. It uses those persons with AD listed as any of the 20 conditions of mortality coded on the death certificates, compared to all other causes of mortality in decedents over 60 by age and sex. The underlying cause of mortality uses the 113 cause of death recode. The twenty conditions of mortality do not have recodes. The analysis of mortality by AD as one of the twenty conditions on the death certificate follows Hoyert's (1996) recommendation and combines Alzheimer's disease with the other causes of dementia, including the ICD-9 categories AD (ICD-9, 331.0), Senility (ICD-9, 797), Senile and presenile organic psychotic conditions (ICD-9, 290), and Other cerebral degenerations (ICD-9, 331.1-331.9). The third treatment compares AD as a dummy variable, scored 1, to all decedents over 60 whose cause of mortality is any of the chronic and degenerative diseases.

Six independent variables are used: age, sex, race, ethnicity, marital status, education level and whether or not the decedent lived in a metropolitan area.

1. Age is measured in five-year periods beginning with decedents age 60-64, 65-69, 70-74, 75-79, 80-84, 85-89, 90-94, 95-99, 100-104, 105-109, 110-114, 115-119, 120+.
2. Sex is a dummy variable coded 1 if male, and 0 if female.

3. Racial categories compiled from the death certificates are Black, White or Asian. Racial categories are analyzed as a dummy variable for each racial group.
4. Ethnic categories provided by NCHS are Non-Hispanic White, Non-Hispanic Black and Hispanic. Ethnic categories are also analyzed as a dummy variable for each group.
5. Marital status categories are obtained from NCHS are Married, Single-never married, Widowed and Divorced. These are analyzed using dummy variables.
6. Education levels used are elementary school, middle school, high school and college, and are also analyzed as dummy variables.
7. Metropolitan status categories used are either metropolitan or nonmetropolitan.
8. Place of death categories are obtained from NCHS; outpatient, residence, inpatient, doa and nursing home.

The literature review noted the research gap in mortality research pertaining to the oldest old, i.e. those over age 85. As the population ages, decedents in this age group are increasing. By compiling five years of data there are sufficient numbers for analysis in the oldest age group. An example, using data from 2001 of the 1,934,858 decedents over 60 in 2001, 18,489 decedents were over age 100. By combining five years of data approximately 80,000-100,000 decedents are over age 100. These numbers allow for statistical analysis of the oldest old, those decedents in the 90's and 100+. These death certificate data will also be used to study whether the trajectory of mortality by AD continues to escalate with age, stabilizes or even decreases, as either an underlying cause of mortality or as one of the twenty conditions on the death certificate.

## Methodology

There will be several methodologies used in this dissertation. The first is a descriptive analysis of causes of mortality among the aged decedents. Descriptive analysis of numbers and percentages is used to show mortality differences by the dependent variable of cause of mortality as well as age, sex, education level, marital status, race and ethnicity. Second, because the dependent variable in the death certificate data is dichotomous logistic regression will be used. In this case a logistic regression model is used to estimate the odds of dying of Alzheimer's disease versus other causes of mortality. Third, because with large data there is an increased likelihood of significance, a Bayesian Model will be used to determine fit of model since because conventional statistical tests may err in the rejection of the Null hypothesis even when intuitively and theoretically it should be accepted.

### Logistic Regression

Long and Freese 2003) have noted that with a binary or dichotomous dependent variable, the outcome of  $Y$  has two usual forms; 1 or 0. 1 is interpreted as a yes or "success" and 0 is interpreted as no or "failure". The  $Y$ 's mean is the proportion of times it takes the value of 1.

$p = P(Y = 1)$ , or,  $p = P(\text{"success"})$

Thus, logistic regression allows one to estimate this probability  $p$  and determine the factors, i.e., independent variables the influence its value.

Hamilton (1992) recommends the logit model because when one has a dichotomous dependent variable, such as one either dies of AD or of other causes.

Logistic regression requires some of the same assumptions as OLS: unbiasedness, efficiency and normality, and large sample size. The specific assumptions are:

1. The model is specified correctly. No important variables are omitted and no extraneous variables are included. Also, **X** variables are measured without error.
2. The cases are independent.
3. None of the **X** variables are linear functions of the others. As we know perfect multicollinearity makes estimation impossible; strong multicollinearity makes estimation imprecise.
4. Influential cases also present problems for logit regression, as they do for OLS.
- 5 Sample size, should exceed 100, not only overall sample size but also on the number of cases with a given combination of **X** and **Y** values (Long and Freese 2003).

Logistic regression has other similarities to OLS regression. A goodness-of-fit test such as model chi-square is available in logistic regression is the “likelihood ratio chi-square” statistic, also called LR chi-square. It’s formula is:

$$\mathbf{LR\chi^2 = -2(Log\ likelihood\ at\ iteration\ of\ 0 - Log\ likelihood\ at\ final\ iteration)}$$

Logit coefficients correspond to b coefficients in the linear regression equation and standardized logit coefficients correspond to beta weights. In OLS regression, which assumes the distribution of the errors is normal, the residual sum of squares for a fitted model is proportional, i.e., similar to the log likelihood at final iteration for the logistic model. Following these assumptions the  $-2 \log (L_0)$  is analogous to the total sum of squares in OLS (TSS), and the  $-2 \log (L_1)$  is analogous to the residual sum of squares in OLS (RSS).

Another similarity between OLS and Logistic regression is the goodness of fit function of  $R^2$  in OLS and known as **Pseudo  $R^2$** , or as  $R^2_L$  or the “likelihood ratio index.” in Logistic regression,  $R^2_L = -2 \log (L_0) - [-2 \log (L_1)] / (-2 \log (L_0))$ . The **Pseudo  $R^2$**  is used as a rough approximation of how the model fits the data, ranging from 0, when the predictors are completely unrelated to the dependent variable, to 1.0, when a model is fitted that allows perfect prediction. However, that **Pseudo  $R^2$**  does not have the "explained variance" interpretation of the  $R^2$  in OLS.

In logistic regression the **z** statistic, also called the **t**-test or a quasi-**t**-test, is used to indicate statistical significance and is similar to the **t**-statistic in OLS. The **z** is the coefficient divided by its standard error, or  $z = b/se$ . The **z** test statistic is considered to have an asymptotic t-distribution, and may be. If the **z** statistic is greater than 2.0, this means that the logit coefficient is at least twice the size of its standard error. This usually results in a probability value of .05 or better, indicating that the logit coefficient is statistically significant (Hamilton 1992).

Logistic regression also allows for a more understandable interpretation of a relationship between the dependent and independent variables. In logistic regression the maximum likelihood estimation can be transformed into the natural log of the odds of the dependent variable for easier interpretation in terms of odds ratios. The odds ratio or  $\Omega$  may be calculated directly by taking the antilog (that is,  $e$  to the power) of the logit coefficient. Poston (2003) notes the odds ratio contains the same information as the logistic regression coefficient or the probability and provides the same ordering of the **X** variables, from strongest to weakest, as the logit coefficients.



### **Bayesian Model**

An issue of using large data sets is of an increased likelihood of significance based on the large numbers. Raftery (1995) recommends the use of a Bayesian Model in social research involving large N's because conventional statistical tests may err in the rejection of the Null hypothesis even when intuitively and theoretically it should be accepted. Accordingly the BIC model, recommended by Raftery for use in large data sets is used. Used with logistic regression, as a rule of thumb, a BIC of 0-2 is weak, 2 - 6 is moderate, 6 - 10 is strong, and over 10 is very strong. (Long and Freese, 2001, 86).

Chapter III has presented the data and methodology of this dissertation. Chapter IV will present the data analysis of the NCHS multiple cause of death files. Chapter V will present the summary, implications for policy and suggestions for future research.

## **CHAPTER IV**

### **RESULTS**

This chapter presents the major results of this dissertation. It consists of an analysis of data from decedents aged 60 and over using the National Center for Health Statistics Mortality Files for the five years of 1998-2005. The analysis examines these likelihoods: 1. the likelihood of dying of AD as the underlying cause of mortality; 2. the likelihood of dying of AD as any of the twenty conditions of mortality; and 3. the likelihood of dying of AD compared to only those who die of chronic and degenerative diseases. The hypotheses and the proposed direction of the relationships are presented first. Next is a general description of the frequencies of the variables. Then the results of the logistic regressions are presented and discussed. All tables depicting results are placed in Appendix G, and referenced in this chapter.

#### **Hypotheses**

A brief listing of the hypotheses and the predicted direction of the relationships between the variables will be introduced before presenting the descriptive statistics and logistic regressions. Please refer to Chapter II for a detailed description of the variables, how they are measured, as well as the complete hypotheses. The projected relationships between the variables and the directions of each hypothesis which will be explored using logistic regression are:

1. Age and AD up to age 90 +
2. Past age 90 and AD -
3. Male and AD –
4. Education and AD-

5. Blacks age 80+ and AD-
6. Hispanic populations:
  - a. Cuban and AD +
  - b. Mexican and AD-
7. Nursing Home residence and AD+
8. Singles and AD+

### **Descriptive Analysis**

The advantage of using a five year composite of mortality data is that there are sufficient numbers for analysis of the oldest old. Descriptive statistics presented here illustrate the epidemiological transition in the United States, with increased longevity and mortality due to chronic and degenerative diseases of the 12,009,528 decedents in the years 1998-2002. Of these decedents 9,643,607 were over age 60. There were three versions of AD used. The descriptive analyses use AD and AD as any cause along with a comparison of all causes. The descriptive analyses show similar patterns in all variables and all ages.

Table G.1.A, Appendix G, describes the top causes of mortality by five year aged 60 and above. AD is the fifth leading cause of mortality; with 313,013 dying of AD, though these are only 3.2 percent of the decedents aged 60 and over. This table shows the doubling effect, with AD increasing as the population ages. The numbers drop significantly after age 80. The only age category that was dropped from the logistic regressions due to small numbers of individuals in the category was the 105+ aged decedents, with only 492 decedents reported to be 105 or over.

Figure G.1, Appendix G, visually presents the mortality occurrence by age, all causes, AD and AD as any condition. Note that mortality by all causes, the more elevated curve, clearly accelerates dramatically until aged 80 to 84 when, as the cumulative effect of mortality peaks, begins to decline revealing a rectangularization. The two lower lines representing Mortality by cause of AD and AD as Any Condition follow similar paths. Together they demonstrate almost a Gaussian curve with the majority of deaths by AD and AD as any condition in the decedents in their 80's. Both have similar patterns of gradual increases in the numbers of decedents beginning with decedents in their 60's and peaking for decedents in their 80's and then declining, ending over age 105. This would appear to give support to Hypotheses 1 and 2.

Table G.1.B, Appendix G, shows the frequency distribution in five year periods of decedents over the age of 60 grouped by ethnicity. The last row of the table illustrates the increased mortality by age. The mortality curve is evident: between ages 60 to 64 there were 675,371 decedents; between ages 65 to 69 there were 913,939 decedents; between ages 70 to 74 there were 1,296,730 decedents; between ages 75 to 79 there were 1,671,023 decedents; between ages 80 to 84 there were 1,822,068 decedents. The frequency begins to decline to 1,667,028 in decedents aged 85 to 89; 1,085,794 decedents between ages 90 to 94; 420,254 decedents between ages 95 to 99; 8,158 decedents between ages 100 to 104 and 492 decedents ages 105+.

Figure G.2, Appendix G, provides a picture of changes in the population by ethnicity as the population ages. Note that from ages 60 to 80 there is a decrease in the percentages of mortality among both Hispanic and NonHispanic Black decedents and a slight increase for NonHispanic Whites. Beginning with decedents aged 90 and over,

there is an increase in the percentages of NonHispanic Blacks and, to a lesser extent Hispanics, with a decrease in the percentage of NonHispanic Whites.

Table G.1.B, Appendix G, provides additional data about the frequency and the percent of decedents by ethnicity and age, revealing racial disparity in the aging population. Among the younger decedents, aged 60 to 64, 76 percent were NonHispanic Whites and 16 percent NonHispanic Blacks; this increases to 79 percent for NonHispanic Whites and 14 percent for NonHispanic Blacks between ages 65 to 69. This ethnic and racial disparity continues until it peaks between the ages 90 to 94 with 89 percent NonHispanic Whites and 7 percent NonHispanic Blacks. Then, between the ages 95 to 99, there is a slight decline with 88 percent NonHispanic Whites and 7 percent NonHispanic Blacks. This becomes more pronounced among the oldest ages. Between the ages 100 to 104 there were 78 percent were NonHispanic Whites and 18 percent were NonHispanic Blacks. At ages 105+ 42 percent were NonHispanic Whites and 44 percent were NonHispanic Blacks. It should be noted that death certificate data on age at death are provided by the decedents' informants and, as is discussed in Chapter II, the validity of age reporting is an issue. However, the informant shown by age of decedent indicates that among the oldest old the Black/White crossover is evident.

Table G.1.C, Appendix G, lists ethnic frequency by age of the 313,103 decedents with AD along with the 486,718 decedents with AD as any condition of Mortality. This table enables one to compare the frequency by cause of AD or AD as any condition and all causes by the three ethnic groups used in the logistic regressions. Consistent with the literature AD appears to be more prevalent among the NonHispanic white population. Although 85 percent of those over age 60 are NonHispanic Whites, 92 percent of the AD

decedents and 91 percent of the AD as any condition are NonHispanic Whites. Comparatively, NonHispanic Blacks make up 9 percent of the over 60 population and only 5 percent of AD decedents and 6 percent of those with AD as any cause. The Mexican decedents comprised 2 percent of the aged 60 + decedents and 1 percent of the AD decedents and 1 percent of those with AD as any condition. The other Hispanic groups had less than 1 percent of the decedents over age 60 and similar small percentages of mortality by AD. Although this table presents the frequencies and percentages for both race and ethnicity, the low numbers in the Hispanic categories required that race and ethnicity be collapsed in the logistic regression, as Hispanic, NonHispanic White, and NonHispanic Black.

Table G.1.D, Appendix G, presents the frequencies of mortality by sex so that one can compare mortality by all causes, AD, and AD as any cause by age. This table shows males dying in greater numbers at earlier ages. Females have increased numbers of mortality by AD; 302 thousand die of AD as any cause versus 184 thousand males. Not only do more females die aged 80-89 when there is the greatest risk of dying of AD, females also have an increased percentage of mortality by AD and AD as any cause at all ages.

The next two tables, Tables G.1.E and G.1.F, in Appendix G, show marital status by age and cause. The married category is the largest, and declines with the mortality curve. The widowed category is interesting in that 55 percent of those who die of AD are widowed compared to 46 percent of those who die of other causes. Lower percentages of decedents with AD or AD as any condition are divorced: 5 percent of both AD and AD as any condition decedents compared with 9 percent of other causes.

Tables G.1.G and G.1.H, Appendix G, show the same trend of similar frequencies among the two versions of the dependent variables, AD as the underlying condition of mortality, and AD as any of the conditions. These tables are especially interesting in that the literature suggests that higher education protects against AD. However, these data indicate a larger percentage of decedents with high school and college education dying of AD and AD as any condition than of other causes. Of those AD decedents 47 percent have a high school or 9 to 12 years of education compared to 50 percent of decedents of all causes; the results for AD as any condition are similar. Those with college education, 13 to 16 years of education, have similar percentages. It should be noted that the education level data may have problems because they are supplied by informants with no verification attempted. Education levels attained are retrospective data, with education levels attained possibly 40 to 60 years prior to obtaining the death certificate data. Data validity could be a problem. Finally, the literature suggests that productive and mentally challenging activities are protective against AD. Education, usually attained in early life, may not be related to whether the decedent continued with productive and mentally challenging activities in later life.

Table G.1.I and G.1.J, Appendix G, differentiate the decedents by metropolitan residence. These data indicate that there is very little difference between those who live in metropolitan or nonmetropolitan areas. About 76 percent of all the groups of decedents are metropolitan residents. There are two possible explanations for this lack of variation. First, there is no difference in the incidence of AD by metropolitan residence. Second, there is a universal medical acceptance of AD in metropolitan and non metropolitan areas

and thus no difference in the diagnosis of AD between the two areas. Metropolitan status is included in Model 5 of the logistic regressions; however it was not significant.

Table G.1.K and G.1.L, Appendix G, depict the place of death by cause of AD or AD as any condition compared to dying of other causes. The most obvious difference is in the area of Nursing Home residence. Half of AD the decedents are nursing home residents compared to 20 percent of those with other causes. The AD as any condition decedents follows a similar pattern; 47 percent are nursing home residents compared to 20 percent of those dying of other conditions. Also supporting the notion of AD being a chronic condition is that whereas 30 percent of decedents of other causes are hospitalized inpatients, only 11 percent of those decedents of AD and 14 percent of those decedents of AD as any condition die as hospitalized inpatients. Finally, low percentages of those with AD are likely to die while living in their own home, i.e., 13 percent of those with AD and 12 percent of those with AD as Any of the Conditions, compared to 18 percent of those with other causes.

### **Logistic Regressions**

This section discusses the results of the logistic regression models used and the extent to which they support the hypotheses. Logistic regressions use the three versions of the dependent variable in five nested models with each model including an increased number of independent variables. Version one, Tables G.2.A to I, Appendix G, depicts AD as the underlying cause of mortality, a dummy variable compared to dying of all other causes. Version two, Tables G.3.A to I, Appendix G, depicts AD as any of the conditions of mortality, a dummy variable compared to dying of any other cause. Both of these visions were used to address questions of coding consistency and of multiple



conditions of mortality. In all cases the logistic regressions using versions one and two provided consistent results, showing that mortality by AD functions in a similar manner despite whether AD is the underlying cause of mortality or one of the conditions of mortality. Version three, Tables G.3.A to I, Appendix G, depicts AD the as underlying cause compared to dying of any other chronic condition; this model allows one to look at AD compared to only chronic causes. Each Table is further divided into A to J subsets including Models 1 to 5, using only those decedents in each of the specific five year age groups. Models 1 to 5 present a nested approach in that one is looking at mortality by AD only for those decedents in the five year group, by an increasing number of independent variables in the five models. Model 1: Sex; Model 2: Sex and Ethnicity; Model 3: Sex, Ethnicity and Marital Status; Model 4: Sex, Ethnicity, Marital Status, Educational level and Metropolitan residence; and finally the full model, Model 5: Sex, Ethnicity, Marital Status, Educational level, Metropolitan residence and location at time of death. The logistic regression results depict the coefficients and percent change in odds ratio with measures of significance and model fitness. Since the models are inclusive of nine age periods each table will have nine sections, one for each age period.

The remaining tables in Appendix G include additional analyses necessary for hypothesis testing: Table G.5.A (Coefficients and Percent Change in Odds Ratio AD as the Underlying Condition in Decedents Aged 60 to 105+, United States 1998-2002); Table G.5.B (Coefficients and Percent Change in Odds Ratio AD as Any Condition in Decedents Aged 60 to 105+, United States 1998-2002); Table G.5.C (Coefficients and Percent Change in Odds Ratio AD as Underlying Condition in Decedents Aged 60 to 105+, Mexican and Cuban Comparisons, United States 1998-2002) and Table G.5.D

(Coefficients and Percent Change in Odds Ratio AD as Underlying Condition in Decedents Aged 60 to 105+, by Ethnicity and Age, United States 1998-2002).

### **Bayesian Measures of Model Fit**

Bayesian Measures of model fit are used for evaluating the models, BIC and BIC' are used to compare the models in this analysis. Long and Freese (2006) note that BIC is defined as  $BIC_k = D(M_k) - df_k \ln_k$ . The more negative the BIC the better the fit. There is also BIC' based on the LR chi-square with  $df'_k$ . BIC' is defined as  $BIC'_k = -G^2(M_k) - df'_k \ln$  (Long and Freese, 2006, 112). The more negative the BIC' the better the fit. Long and Freese note that form selection is a matter of convenience as both give the same information. With both forms, when considering selection of the best fit of multiple models, one should compare the two,  $BIC_1 - BIC_2$ . When the result is  $<0$  one should use the first model; when the result is  $>0$  one should use the second model. The rule of thumb for the absolute difference between the models is: 0-2 denotes weak support, 2-6 positive; 6-10 strong;  $>10$  very strong. The full model i.e. Model 5, was used as the standard for model comparison. Note the next to the last row of the tables (2.A to I; 3.A to I and 4.A to I) provides the model comparisons using BIC. Although models of fit, including BIC, do tend to accept the most complete model as the best fit, the full Model or Model 5 is used as the preferred model according to the BIC analysis. There are three exceptions to Model 5 as the best fit: the subsets addressing decedents aged 100 to 104; in Appendix G, Tables G.2.I and G.4.I reflect that Model 1, sex alone was preferred and Table G.3.I shows that Model 4 was the preferred model.

For ease of interpretation the percent change in odds ratio will be used although both the coefficients and percent change in odds ratio are included in the tables in the

appendix. Refer to Table G.2.A for an explanation of the use of the percent change in odds ratio. Male is a dummy variable, compared to females. The second column, row 1, shows that “Male” has a logit coefficient of -0.272. This may be exponentiated to an odds ratio of 0.762. We next subtract 1.0 from this value and multiply by 100 to yield the percent change in the odds ratio. This value of -23.8 (shown under the logit coefficient) means that in Model 1 the odds of males, compared to females are 24 percent less of dying of AD holding all other variables constant. This result is for decedents age 60-64. The percent change in odds ratio is used as the preferred interpretation as it allows for a proportional representation which gives a clearer presentation of the change in the coefficients.

The level of significance is not really an issue when one has large numbers of cases. Note in the last row of Tables G.2. A to I, G.3.A to I and G.4.A to I the level of significance is  $*=P<.01$  and  $**=P<.05$ . Only significant findings will be noted in the narrative below. Unless specified the level of significance is  $P<.01$ .

## **Hypotheses**

### *Hypothesis One and Two*

Hypothesis one predicts that age and AD, up to age 90, will have a positive relationship whereas hypothesis two predicts that past age 90, age and AD will have a negative relationship. The results support hypotheses one and two. These hypotheses have been addressed in the descriptive data in Figure G.1, Appendix G. Table G.5.A, Appendix G, depicts the logistic regressions results comparing those decedents with AD with the standard of decedents aged 60 to 64. Decedents at age 65 to 69, compared to decedents aged 60 to 64, had 2 percent less odds of mortality of AD, holding all

variables constant, whereas in all other age categories there was an increased likelihood of mortality by AD. This trend continues peaking with decedents aged 90 to 94 having a 512 higher percent odds ratio. Then there is a slight decrease for decedents aged 95 to 99; similar to decedents aged 85 to 89. This continues with the oldest ages.

Table G.5.B, Appendix G, shows the logistic regression results comparing those decedents with AD as any condition. They follow the trends of AD. However, as would be expected, there are increasing percentages in the odds ratio of having AD as any condition of mortality that peak and then drop as already shown in Figure G.1, Appendix G. This Gaussian shaped curve is evident throughout the analyses with changes in mortality by AD following these same trends throughout. If one uses the path of mortality by AD, supported by the literature review in Chapter II, that AD is chronic and gradually progressive, with mortality following from five to twenty years after onset, the peak years of onset of AD may be from sixty to eight-five years. This would support a critical onset age theory, with less likelihood of the onset of AD in the oldest ages.

### *Hypothesis Three*

Hypothesis three predicts that males, compared to females, will have a decreased likelihood of dying of AD. The results support hypothesis three. In Table G.2.A, Appendix G, one first notes the relationship of sex to mortality by AD depicting the odds of dying of AD limited to those decedents between ages 60 to 64. As the nested models increase in the number of variables the direction of the male decedents being less likely to die of AD remains, though there is some variation. In Model 5, holding all variables constant, the odds of males dying of AD are 21 percent less than for females. Again holding all other variables constant, this negative relationship between male sex and AD

continues as a significant variable with males dying of AD decreasing by a percent change in odds ratio ranging from -16 percent at age 65 to 69 to -49 percent for those aged 100 to 104,. Tables G.3.A to I, Appendix G, show that the negative relationship between male sex and AD as Any Condition follows the same general trend. Throughout these tables one also notes that their coefficient peaks in mortality in the 80's owing to the increased longevity of females. Females were more likely to die of AD in the advanced ages.

Tables G.4.A to I, Appendix G, depict the relationship between dying of AD versus dying of chronic and degenerative conditions. The negative relationship between male sex and AD follows the above trend. In the full model males dying of AD, compared to dying of other chronic diseases, decreases by a percent change in odds ratio ranging from -18 percent for decedents aged 70 to 74 to -50 percent for decedents aged 100 to 104.

#### *Hypothesis Four*

Hypothesis four predicts that education will be negatively related with mortality by AD. Those with higher education should be less likely to die of AD. This hypothesis is not supported. Please refer to the possible reasons listed in the descriptive section above.

Tables G.2.A to I, Model 5, Appendix G, depict the relationship between AD and both high school and college education with elementary education as the reference. Table 2.A indicates for decedents aged 60 to 64 the high school education variable is not significant, and those with college education have an increased risk in the likelihood of mortality by AD. Table G.2.B, Model 5, Appendix G, shows for decedents aged 65 to 69 the odds of dying of AD are 12 percent higher for those with high school education, and

66 percent higher for those with college education. This relationship continues in the same direction, with a significant positive relationship through Tables G.2.H, Appendix G, ages 95 to 99. Only Table G.2.I, depicting ages 100 to 104 shows no positive significant relationship between dying of AD and college education and a negative relationship between dying of AD and high school education. Tables G.3.A to I follow the same pattern shown in Tables G.2 A to I, depicting the relationship between AD as any condition. Table G.4.A to I, dying of AD compared to other chronic causes, show similar trends.

#### *Hypothesis Five*

The fifth hypothesis is that NonHispanic Blacks, who survive beyond age 80, will have a decreased mortality by AD compared to NonHispanic Whites. As presented in Figure G.2, Appendix G, this hypothesis is confirmed. Among all decedents over aged 60, NonHispanic Blacks are less likely to die of AD than NonHispanic Whites. The logistic regressions also show decreased mortality by AD for NonHispanic Blacks in all three versions of AD as the dependent variable. Tables G.2.A to I, Appendix G, depict a decrease in the percent change in odds ratio of mortality by AD for NonHispanic Blacks, compared to NonHispanic Whites. This ranged from a decrease of 59 percent in Table G.2.A, Model 5, for aged 60 to 64, to a decrease of 5 percent in Table G.2.I, Model 5, among decedents aged 100 to 104. Tables G.3.A to I, Appendix G, depicting AD as any condition of mortality, and Tables G.4.A to I, Appendix G, depicting AD compared to only chronic causes of mortality, show, roughly speaking, the same relationships.

*Hypothesis Six*

It is recognized that there is no one Hispanic population. Then the relationship of Ethnicity and AD should involve multiple analyses. Because the Cuban population in Florida is an older population with positive socioeconomic support it was hypothesized that they would be more likely to die of AD. The hypothesized direction between Mexican and AD should be negative as the Mexican population, particularly in the five Southwestern States, Texas, California, New Mexico, Arizona, and Nevada, is a younger population with fewer socioeconomic advantages. Table G.5.C, Appendix G, provides partial support for these hypotheses. Mexicans, compared to other ethnic groups, have a lowered AD mortality, holding all other variables constant. Mexicans who live in the SW have a 43 percent lower AD mortality. Cuban AD mortality is not significantly different from the comparison group of NonHispanic Whites. This holds for Cubans who live in Florida as well as for those who reside in other states.

The issue of Hispanic ethnicity analysis is also addressed in Table G.5.D, Appendix G, which presents mortality by age and ethnicity, separating the Hispanic decedents into Mexican, Cuban, Puerto Rican and Other Hispanic categories. Of the Hispanic ethnic decedents only Mexicans have significantly decreased mortality by AD across all ages. Decreased mortality by AD among Cuban and Puerto Rican decedents becomes significant only at the advanced ages: Cuban decedents at age 85 to 89 and Puerto Rican decedents at aged 75 to 79. It is noteworthy that in this analysis the Hispanic decedents have decreased mortality by AD. The only exception, not significant, is among decedents aged 100 to 104.

Due to low frequencies and the confirmation above that directions of the other Hispanic ethnic groups were similar, all of the Hispanic groups were combined for analyses using the three versions of AD and five models. Combined, as in Table G.2 A to I, Appendix G, the odds of Hispanics and NonHispanic Blacks dying of AD, compared with other causes, also shows the predicted negative relationship. Although the direction is negative for all ages, in the full model, Model 5, the BIC best fit, is significant only for three age groups. Decedents aged 60 to 64 have a decreased odds of mortality by AD of -19. Decedents aged 80 to 84 and 90 to 94 have a decrease of -7 percent.

Tables G.3.A to I, Model 5, Appendix G, depicting AD as any condition, show similar patterns. In Tables G.4.A to I, Model 5, Appendix G, depicting AD compared to other chronic causes of mortality there is a slight decrease in mortality of AD compared to other chronic causes. The data show that while the relationship of Hispanics is negative as hypothesized, for decedents of most ages, mortality by AD does not vary significantly from that of NonHispanic Whites.

### *Hypothesis Seven*

This hypothesis uses a proxy of the increased disability of AD and its accompanying financial burden predicting that AD decedents will have an increased likelihood of a Nursing Home being the place of death. Of all of the variables nursing home as place of death presents the greatest increase. Tables G.2.A to I, Appendix G, show that the odds of nursing home residents, aged 60 to 64, dying of AD increases by 887 percent, holding all other variables constant. This relationship continues in all other aged decedents ranging from a 598 percent increase for decedents aged 65 to 69 to an increase of 38 percent increase for decedents aged 100 to 104.



Tables G.3.A to I, Appendix G, depicting AD as any condition also support this hypothesis. The odds of nursing home residents dying of AD as any condition range from 702 percent among those decedents aged 60 to 64, to 40 percent increase for decedents aged 100 to 104. Tables G.4.A to I, Appendix G, depicting AD compared to chronic causes of mortality, show the strongest support for the hypothesis that there is a positive relationship between nursing home as place of death and AD. The odds of nursing home residents aged 60 to 64, dying of AD compared to dying of other chronic causes increases by 909 percent, this relationship continues in all other ages.

#### *Hypothesis Eight*

This hypothesis expects that singles will be more likely to die of AD compared to those who are not single. The results of this analysis only partially support the hypothesis. This may be due to the small number of singles who survive over the age of 60. If one compares only the married with the single then the direction of the relationship is not as hypothesized. Tables G.2.A to I, Model 5, Appendix G, show that among married decedents, compared to singles, the odds of dying of AD, versus dying of all other causes, increase from 14 percent in decedents aged 60 to 64 to 82 percent in decedents aged 100 to 104. Results shown in Tables G.3.A to I, Model 5, Appendix G, of the odds of dying of AD as any condition, compared to dying of all other causes and the results in Tables G.4.A to I, Model 5, Appendix G, showing the odds of dying of AD, compared to dying of only chronic causes, are all similar.

Conversely, the widowed, have decreased odds of dying of AD. Tables G.2.A to I, Model 5, show that the percent change in odds ratios ranged from -22 percent for decedents aged 60 to 64 to -6 percent aged 90 to 94 (in the older ages the relationships

are not significant). In Appendix G, Tables G.3.A to I, Model 5, AD as any condition and Tables G.4.A to I, Model 5, dying of AD, compared to dying of chronic causes show similar patterns.

The divorced, compared with the single, follow the same trends as the widowed.

### **Conclusion**

This chapter has presented the basic analyses of data for decedents aged 60 and over, using the National Center for Health Statistics Mortality Files from 1998-2005. Both descriptive statistics and logistic regressions were presented to address the hypotheses. Data testing eight hypotheses were presented. The analyses supported six of the hypotheses, and the remaining two had partial support.

Age and AD had a positive relationship up to age 90. Over age 90, age and AD had a negative relationship. Males were less likely to die of AD than females, a negative relationship as hypothesized. The relationship between education and AD was positive with high school and college having an increased likelihood of AD, which was not as hypothesized. NonHispanic Black decedents over age 80 were less likely to die of AD. Data supported the hypothesis that NonHispanic Black decedents over 60 were less likely to die of AD than NonHispanic Whites. Mexicans who lived in the SW as well as all Mexican decedents were significantly less likely to die of AD across all age categories. The combined Hispanic data showed that for decedents of most ages, mortality by AD does not vary significantly from that of NonHispanic Whites. However, the relationship of Hispanics was in the negative direction as hypothesized. Nursing Home as place of death and AD had a strong positive relationship as was hypothesized. Of all of the variables, nursing home as place of death shows the greatest increase in percent change in

odds ratio. Singles were hypothesized to have a positive relationship with AD. The results of this analysis only partially support the hypothesis. The married were more likely to die of AD than the single however the widowed and divorced were less likely to die of AD than the single.

All tables depicting results are placed in Appendix G, and referenced in this chapter. Chapter IV will summarize the results of this dissertation, present policy implications and discuss the need for future research.

## **CHAPTER V**

### **CONCLUSIONS**

This concluding chapter has three sections. The first section is an overview of the findings of the dissertation including the statement of purpose, a summary of data, methodology, and hypotheses. The second section presents the major implications of the research including implications for public policy. The third section is a retrospective analysis of how this work could have been improved specifying questions that have surfaced from these analyses which might be addressed in future research.

#### **Overview and Summary**

The purpose of this research was to explore the dynamics of mortality caused by Alzheimer's disease and dementia (AD) among those over age 60 in the United States. The initial research questions posed were the following: what is the extent of mortality by AD among elderly decedents. Although AD is age related with a doubling effect cited in the literature (Antuono and Beyer 1999, Dalsania 2004, Gambassi et al. 1999, Hoyert 1996, Hebert et al. 2003, Nocera et al. 2003, Pollen 2000) research on AD in the oldest old is limited to small numbers in those over age 90. Researchers (Gao et al. 1998, Hofman et al. 1991 Ritchie and Kildea 1995) have attempted to address the small numbers in AD research of the oldest old through a meta analyses. General findings were inconclusive but suggested that incidence of AD did decrease after age 85.

The primary significance of this dissertation is its large scale. This study was influenced by Carey's (2003) suggestion that if one wants to study longevity and the trajectory of mortality at advanced ages one must have sufficient numbers to have survivors at the end of the curve. Since the literature suggested that even attempts at meta

analysis had limited numbers what was needed was a study with sufficient numbers to address the trajectory of mortality by AD in those over age 90. This dissertation combined five years of NCHS mortality data, with 9.5 million decedents over age 60; 1,085,794 decedents aged 90-94; 420,254 decedents aged 95 to 99; 8,158 decedents aged 100-104 and 492 decedents aged 105+ (refer to Table G. 1.B in Appendix G). Due to the years included, 1998-2002, no period effects are anticipated to affect the results. Although the 9/11/01 mortality is included in this period it did not impact this study as the focus is on the population over age 65 and NCHS data indicates the majority of those who died were between ages 35 to 39 beyond the scope of this study.

As the title of this dissertation implies we are entering an era of global aging. Longevity researchers positing the rectangularization of mortality (Carnes and Olshansky 1993, Olshansky and Carnes 1994, Olshansky et al. 1990, Olshansky et al. 2001, Olshansky 2003) posit that there is a possibility of increased life expectancy as individual causes of mortality are reduced. However, due to comorbidity, future life expectancies will not have the same dramatic increases as in the last century. The opposing argument posed by other researchers (Alburg and Vaupel 1990, Horiuchi and Wilmoth 1998, Manton et al. 1991, and Vaupel et al 1998, Vaupel 2001) posit that we are altering senesce. Life expectancy has been increasing for the past 160 years and life expectancy may reach well over 100. Alburg and Vaupel (1990) project a life expectancy of 100 years for females in developed countries in 2080. Horiuchi and Wilmoth (1998) present three stages of mortality in those who survive past: a deceleration of mortality after age 80, a plateau between ages 80 to 105 and an actual decline in mortality in the highest ages over 110. Since AD is assumed to be age related, what is the relationship between AD

and age at these oldest ages? What is the direction of the relationship between mortality by AD and the variables of age, sex, education level, marital status, metropolitan status and place of death?

The dependent variables were three: AD as the underlying cause of mortality, and AD as any condition of mortality compared to both all other causes, and to other chronic causes among those over sixty years. This research focused on the extent of AD in the elderly population. Chapter IV presented the data analysis testing the following seven hypotheses, and the proposed direction of the relationships among the variables: 1. Age and AD up to age 90 (+); 2. Age and AD over age 90 (-); 3. Males and AD(-);4. Education and AD (-); 5. Blacks over age 80 and AD (-); 6. Hispanic populations: a. Cuban and AD (+); b. Mexican and AD (-); 7. Nursing Home residence and AD (+);8. Singles and AD (+).

This section will summarize the findings by hypothesis, combining one and two as they are both age related hypotheses. There were three versions of the dependent variable, as well as five models. Although three versions of AD were used, this summary will use only the fullest version, AD as any of the conditions of mortality, and Model 5, including all of the independent variables; this was shown to be the best model according to the Bayesian analyses.

### *Age*

The first and most significant finding was the relationship between age and mortality by AD. These findings answer the question raised by the title of this dissertation, showing that although AD is the fifth leading cause of mortality of those over age 60, it is not inevitable with aging. Mortality by AD decreases in the oldest old.

First initially AD does increase with age, beginning at age 60. Descriptive statistics show that AD as any condition of mortality ranges from .7 percent of all causes for decedents aged 60-64 to 7.9 percent of all causes of mortality for decedents aged 90-94.

However as is mentioned above this study was able to address the oldest old. Mortality researchers (Brookmeyer et al. 1998, Brookmeyer et al. 2002, Gatz et al. 2000, Ganguli et al. 2005, Ritchie and Kildea 1995) reviewed in Chapter II indicated a gap in research of the oldest old. Prior studies had either used too limited a sample size of the oldest old or extrapolated findings for the oldest age group from younger ages due to the small numbers of those surviving to the oldest old. One advantage of this dissertation was the large population of 12 million decedents in the years 1998 to 2002. Of these decedents 9.5 million were over age 60. The only age category that was dropped from the logistic regressions due to small numbers of individuals in the category was the 105+ aged decedents, with 405 dying of AD as any condition.

Perhaps the most significant finding was that age and AD had a positive relationship up to age 90. The tables referenced in this section are in Appendix G. The descriptive statistics, shown in Table G. 1.C, show the doubling effect of mortality by AD that is often mentioned in the literature. Four thousand six hundred died of AD as any condition among decedents aged 60 to 64; 11.7 thousand, 65 to 69; 31.6 thousand, of those aged 70 to 74; 72.9 thousand, 75 to 79; 116 thousand, 80 to 84; 127 thousand, 85 to 89. However, age 90 to 94 the numbers began to decline with 85 thousand dying of AD as any condition. This decrease continued as 31 thousand died of AD as any condition aged 95 to 99 decreasing to 5 thousand for age 100 to 104 and .5 of a thousand for aged 105+. These declining numbers of course were due in part to declining numbers of

decedents. Furthermore, a limitation of this study, relying on informants who complete the death certificates, is that those who code mortality in may not adequately code the comorbidity conditions.

The logistic regressions showed this relationship. Decedents at age 65-69, compared to decedents aged 60-64, had a decreased mortality by AD of 2 percent, holding all variables constant. In all other age categories there was an increased likelihood of mortality by AD. Decedents aged 70-74 had an increased percent change in odds ratio of 80.1 percent, aged 75-79 an increase of 221.4 percent, aged 80-84 an increase of 376 percent, and aged 85-89 an increase of 476.7 percent. This peaks with decedents aged 90-94 having an increased percent change in odds ratio of 512 percent. Then there is a slight decrease as decedents aged 95-99 have an increased percent change in odds ratio of 496 percent, similar to decedents aged 85-89. This continues with the oldest ages; aged 100-104 an increase of 292.5 percent and aged 105+ an increase of 86.7 percent similar to the age 70-74.

Interestingly the morbidity literature also shows that the length of morbidity varies by age, with those with early onset-in their 50's or 60's having the longest morbidity of AD, while those who have a later onset, in their 80's having the shortest morbidity of AD, approximately 3 to 5 years (Brookmeyer et al. 1998). Therefore this research suggests that the onset of AD may be age graded with the greatest likelihood of onset being in the years 60 to 89.

These findings should be considered in the light of other research that shows that AD mortality is different in advanced ages. Gambassi and colleagues (1999) found that autopsies of those who die of AD at advanced ages show fewer senile plaques and



neurofibrillary tangles, the key autopsy diagnostics for AD. In the advanced ages those who died of AD also had less likelihood of dying in a nursing home than those dying earlier. This analysis indicates a different presentation of AD in the advanced ages. In most cases the variables did not differ significantly for those over age 100, indicating that among the oldest old there may well be more similarities than differences. For example ethnicity, marital status and nursing home residence did not differ significantly among those diagnosed with AD as any Condition (Table G. 4.I).

### *Sex*

The relationship between male sex and AD mortality was negative as hypothesized. This is the second significant finding as the literature reports conflictual findings about the extent of mortality by AD and whether the increase in mortality by females is due to the increase of females surviving to older ages or an actual increase in females' incidence in AD over males. Table G. 1.C, Appendix G, showed that of the 486,718 who died of AD as any condition of mortality 302, 414 were female and 184,304 male. At the younger ages 60 to 69 there were more males who died of AD as any condition; however this must be considered in the light of greater male morbidity in the younger ages. The logistic regressions in Tables G.3.A to J showed the percent change in odds ratio, the odds of males dying of AD as any condition, holding all other independent variables constant. A negative relationship between male sex and AD is significant with males dying of AD as any condition decreasing by a percent change in odds ratio ranging from -9 percent for decedents aged 65 to 69 to -44 percent for decedents aged 100 to 104. Throughout these tables one also notes that mortality by AD as any condition peaks in the

80's. The difference between the sexes, with increased longevity of females as well as their increased likelihood to die of AD as any condition, is also evident at all ages.

### *Marital Status*

The mortality data shows that in the United States by time of death the majority of the population had been married at one time. The disadvantage of the data is that it did not distinguish how long a decedent had been married, divorced or widowed. Death certificate data also is informant data with no attempt at verification. So, to accurately reflect the benefits or risks of marital status, more data from a more complete source is needed. Data showed 5 percent single decedents of AD or AD as any condition compared to about 6 percent single decedents of other causes. Lower percentages of decedents with AD or AD as any condition are married; about 35 percent married decedents of AD or AD as any condition compared to about 39 percent married decedents of other causes. More widowed die of AD as any condition 55 percent, compared to 46 percent of those decedents of other causes. Lower percentages, approximately 5 percent of both AD and AD as any condition decedents, are divorced compared with 9 percent of other cause decedents. The implication from marital status is that married persons live longer, are healthier, and have a support system to assist the person with AD remain in their home, receiving less expensive care. However, being married did not decrease mortality by AD.

### *Ethnicity*

The relationship between Hispanic ethnicity and AD indicates, as does the research literature, that although there is no one Hispanic ethnic population, the mortality of Hispanics is more similar to Non Hispanic Whites than to NonHispanic Blacks. This

researcher conducted four analyses to address Hispanic ethnicity. The first analysis used the nine categories of ethnicity listed on the death certificates and provided descriptive statistics. The second analysis addressed Cuban differences; those Cubans who lived in Florida were distinguished from those who lived in other locations. There were no significant findings. The third analysis addressed Mexicans who lived in the five southwestern states and those who lived in other locations. The combination of both Mexicans who lived in the five southwestern states and the total Mexican ethnic decedents did result in significant decrease in mortality by AD compared to NonHispanic Whites across all age categories. The fourth analyses were AD logistic regressions of Mexicans, Puerto Ricans, Cubans and NonHispanic Blacks compared to NonHispanic Whites by ethnicity and age in five year periods. The significantly different Hispanic analyses focused on Mexicans. Of the other Hispanic decedents only two age groups were significant; Cuban decedents aged 85-94 and Puerto Rican decedents aged 75-94 had significantly decreased mortality by AD compared to NonHispanic Whites.

Next, the Hispanic ethnic groups' combined data were used in logistic regressions. The ethnicity results showed that NonHispanic Whites, compared to NonHispanic Blacks and Hispanics, were most likely to die of AD. Tables G.3.A to J show this relationship. The relationship varies inversely with age, ranging from a significant -8 decreased percent change in odds ratio for NonHispanic Blacks aged 95 to 99 to a -46 percent change in odds ratio between ages 60 to 64. For decedents of most ages mortality by AD does not vary significantly from that of NonHispanic Whites; however, the relationship of Hispanics was in the negative direction as is hypothesized.

My research also found changes in population by ethnicity as the population ages. From ages 60 to 80 there is a decrease in the percentages of both Hispanic and NonHispanic Black decedents and a slight increase in the percentages of decedents who are NonHispanic White. Beginning with decedents aged 90 and over there is an increase in the percentages of NonHispanic Blacks and, to a lesser extent Hispanics, with a decrease in the percentage of NonHispanic Whites. This would suggest support for the theory that minority longevity is due to their being a homogenous healthy population, with only the fit surviving to older ages while the NonHispanic White population is heterogeneous; for them due to majority advantages, the weaker, with genetic disease predispositions can survive longer. According to this theory the NonHispanic Whites would be more susceptible to the chronic and degenerative diseases than the minorities.

Among the younger decedents aged 60 to 64, there were 76 percent NonHispanic Whites and 16 percent NonHispanic Blacks. This increased to 79 percent NonHispanic Whites and 14 percent NonHispanic Blacks for decedents aged 65-69. Peaks occurred with decedents between ages 90 to 94, with 89 percent NonHispanic Whites and 7 percent NonHispanic Blacks. Next, between ages 95 to 99, there is a slight decline; 88 percent are NonHispanic White and 7 percent of the decedents are NonHispanic Black. In the oldest ages there appears to be evidence of mortality crossover. Decedents aged 100 to 104 were 78 percent NonHispanic White and 17 percent NonHispanic Black. For decedents aged 105+ there were 42 percent NonHispanic White and 44 percent NonHispanic Black.

### *Education*

My research only found the predicted negative relationship between mortality by AD and higher education among decedents aged 60 to 64. In all other ages there are larger percentages of decedents with high school and college education dying of AD and AD as any condition than of other causes compared to those with elementary education. The descriptive data show 38 percent of those decedents due to AD as any condition have a high school education compared to 34 percent of those decedents of other causes; and 26 percent of those decedents due to AD as any condition have a college education compared to 23 percent of those decedents dying of other causes.

Tables G.3.A to I, Appendix G, depict the relationship between AD as any condition and education. The hypothesized negative relationship was only present in one age group. The odds of dying of AD as any condition for decedents aged 60 to 64 decreased by 35 percent in Model 5 for those with college education. Significant positive relationships between AD as any condition and high school and college education compared to elementary education continue through decedents aged 95 to 99. In Table G.3.I, it was shown that for decedents aged 100 to 104, there is a negative -34 percent relationship between dying of AD as any condition and high school education level, and no significant relationship with college education.

### *Place of Death*

Nursing Home as place of death and AD as any condition had a strong positive relationship as was hypothesized. Of all the variables, nursing home as place of death presents the greatest variation with mortality by AD and other causes. The results in Table G.1.M, Appendix G, indicate that 47 percent of decedents with AD as any

condition died in a nursing home, that is, of the 486,718 decedents of AD as any condition, 226,742 died listing nursing home as place of death. The logistic regression percent change in odds ratio ranged from an increased 702 percent change in odds ratio for decedents aged 60 to 64 to 40 percent change in odds ratio for those aged 100 to 104. Again this depicts that for decedents of AD as any condition, younger decedents, who according to the literature have longer morbidity, are more likely to reside in nursing homes and receive the more expensive type of care.

Also supporting the notion of AD being a chronic condition is that whereas 30 percent of decedents of other causes are hospitalized inpatients, only 11 percent of those decedents of AD and 14 percent of those decedents of AD as any condition die as hospitalized inpatients. Finally, low percentages of those with AD are likely to die while living in their own home. Only 12 percent of those with AD as Any Condition died in their own residence compared to 18 percent of those with other causes.

### **Implications**

The implications section includes two subsections. The first is a description of the population who will be at risk for AD. Although this analysis of cause of death by AD showed that there may be a deceleration of mortality by AD in the oldest old the greater impact to our systems is the increase of survivors to the critical ages for mortality by AD, aged 80-90. Second are implications for policy development and services for those with AD in the existing Medical, Hospital and Prescriptive medication programs; Hospice and Home Health programs and Long Term Care programs.

### *Demographic Implications*

In 2005 life expectancy in the U. S. is now 77 and three nations have life expectancies over age 80: Japan's is 82, Iceland's is 81 and Sweden's is 81. When the U. S. Social Security System was founded in the 1930's the life expectancy was 62; thus the years of receiving benefits were minimal. Policy planning should be directed toward a baby booming population who will likely have a life expectancy closer to 85 than to the 62 anticipated by the founders of the Social Security system in the U. S.

Currently the U. S. system is addressing the impact of the aging of the baby boomers on the Social Security System and the health care system. This dissertation raises hope in that although we anticipate greater numbers who will experience the increased mortality by AD in the peak mortality years from 80 to 90, there is decreased mortality by AD in those who survive past 90. This goes along with longevity research that reveals an aging but healthier population, with fewer disabilities. If, as current media suggests, 70 is the new 50 then will 100 be the new 70 in 2025? If the aging population is healthier then they may be able to be more productive, allowing for a different presentation of the dependency ratio. The wealth distribution of the aging population of baby boomers is also different in that they may have different resources than other cohorts including home ownership, stored wealth, and private retirement policies; altering their need for Social Security.

Continued demographic analysis of the aging population is vital to planning for this new stage for baby boomers. The social and political systems of the past fifty years have been altered by this large cohort. The major educational, marketing and financial systems have responded to the population pressures as the boomers have gone through

life stages. The first of the cohort is now aged 60, and will soon be entering retirement, and also the stage of chronic and degenerative diseases. The current retirement system was designed with the expectation that there would be a large work force supporting a small retirement cohort with workers living only a few years into retirement. This aging of the population, owing to decreased fertility and increased life expectancy, will necessitate revamping our retirement system. Although the age at retirement has been modified, the dependency ratio will be a critical part of planning.

The United States is twentieth in life expectancy in the world. Other nations with longer life expectancies will thus be dealing with the increased population over age 85 years prior to the U. S. Policy planners should pay close attention to the health and retirement systems of these more mature populations so that practical aspects can be used in revamping the U. S. system.

The changing population structure indicates that with an increase in females over age 80, and their greater likelihood of mortality by AD, we may have a proportional increase in AD. The increased life span of females projected by the U.S. Census Bureau from 2000-2050, for women over age 80 is significant. Table A.1, Appendix A, provides projections. The U. S. Census Bureau shows that in 2000, of the 9,251,968 persons over the age of 80, 6,158,663 were female or 4.3 percent of the female population. However, midrange projections for 2050 are that 33,696,359 or 8 percent of the 420,080,587 population will be over age 80. Females over 80 will number 20,406,847, or 9.6 percent of the U. S.'s 213,408,448 female population. Dalsania (2004) projected that by age eighty-five, 30 to 50 percent of the population will develop dementia. This study found the ages 85 to 89 had the greatest likelihood of dying of AD. Therefore we will have a



larger portion, almost double the present number, reaching the critical age of mortality by AD.

Not only will females have greater rates of AD, the literature indicates that males and females also have different presentations of AD. Researchers (Dodge et al. 2003, Ganguli et al. 2005, Hoyert 1996, Katzman and Bick 2000, Larson et al. 2004) concur that the age of onset and the duration of survival differ by sex. Females are diagnosed earlier and live longer, up to twice as long after AD diagnosis, with increased disability in comparison to males. Lapane and colleagues (2001) found that females who have AD have more symptoms with a faster decline and are thus more likely to live in nursing homes and less likely to have family caretakers.

The financial costs of AD are beyond the scope of this study. However Hay and Ernst (1994) noted that total costs of Alzheimer's disease were \$173,932 per case in 1991 dollars. In the five year period of this study there were 486,718 who died with a diagnosis of AD as any condition. Since that time the population at risk for AD has grown, and the costs of nursing home care and prescription medications have grown as well. This increase in cost is accompanied by a decrease in those who will pay. The dependency ratio, Table A.2, Appendix A, is decreasing with the 65+ dependency ratio, comparing those over age 65 to those in the working years of 15-64, in the US in 2000 was 18.75, in 2050 it is projected to be 34.57.

### **Implications for Policy Development for Services**

Since AD is a chronic condition requiring years of care, ranging from 5 to 20 years, there are two ways to manage expenses: to provide the needed care in a less expensive manner, and prevent or delay the onset of AD.

*Provide Care in Less Expensive Manner*

There are four service systems that currently provide services to those with AD: Hospital Care, Prescriptive Medications, Hospice and Home Health, and Long term Care/Nursing Homes. This section will suggest that improvements in each of these systems will be necessary due to the large numbers who will be requiring services in future years in the U.S.

The expense of AD as the fifth leading cause of mortality in those over 65 in the United States is related to the lengthy morbidity. AD has morbidity from 5 to 20 years. Policy planning must include both development of treatments to cure or delay the onset of disability from AD, and work to develop less expensive ways of care and treatment during the years of morbidity. The research reported in this dissertation used place of death as a proxy for the extensiveness of services provided. The services ranging from least to most expensive are residence, outpatient, (dead on arrival) emergency room, inpatient, and nursing home. The findings were that those who died of AD had a greater likelihood of dying in nursing homes, the most expensive form of care, with about 47 percent of the decedents dying in nursing homes.

While remaining in own residence is the cheapest solution, this study showed that limited percentages of those with AD died while living in their own home. Twelve percent of those with AD as Any of the Conditions died in their own residence compared to 18 percent of those with other causes. Blieszner and Shifflett (1990) found that the quality of life as well as the general health of the demented person was improved by remaining in family care. Both family members who were caretakers and the individual surveyed preferred to live independently or in their residence with assistance. Assistance

needs included: Home repair; Senior Centers; Friendly visitation; Telephone assistance; Home Delivered Meals; Homemaker/Chore services; and Adult Day Care. They agreed that in order to minimize caretaker stress, the care giving family member also needs respite, day care and support groups. If these services can be provided, then more demented family members could remain in the community with their spouses or children providing caretaking with significant cost saving over nursing home care.

Hospital care, in our current system is relegated to diagnostic related groups (DRG's) equating specific diseases to specific treatment courses that can respond to specific treatments in a specified time. DRG's do not allow for the possibility of co-morbidity in a chronic population. Private and Medicare insurance programs limit hospital care to the treatment of diseases that require nursing and physician care. The care of an individual with AD is, for the most part, providing daily living assistance and routine care that do not require a medically skilled practitioner. Because there are no short term treatments for long term chronic illnesses hospitals are not the treatment place for chronic conditions such as AD. Thus our current system directed towards short term treatment of acute symptoms, the DRG costing method, does not work well with AD. Data reflect this as 30 percent of decedents of other causes had place of death as hospitalized inpatients but only 14 percent of those decedents of AD as any condition die as hospitalized inpatients.

While Home Health and Hospice care were not addressed specifically they are part of the outpatient services that would be included on the death certificate. Unfortunately, as is indicated in Table G.1.L and M, Appendix G, only 1 percent of those who died of AD compared to 5 percent of those who died of all causes reported

outpatient care. Home Health services provided through Medicare provides ancillary services to those who are transitioning from hospital care services. Thus there are limits to length of service, rendering those with long term chronic conditions, with some exceptions, not eligible for care. These services would need to be expanded to deal with a chronic population so that full range home health services, Nursing, Dietary, Occupational and Physical Therapy services could be offered on a chronic basis. Those with AD will need care until death, possibly requiring several years of service.

Hospice services also provide full range services for those who prefer to die in their residence, a family member's home, or a hospice palliative care residence. These are also time limited with eligibility restricted to the last six months of a terminal disease. The issue with AD is that it is difficult to determine a six months period of eligibility, one can live for years in a chronic debilitated condition without dying. Therefore both Home Health Care systems and Hospice systems will need eligibility criteria to be modified so that those with from 5 to 20 years morbidity will remain eligible for services enabling them to remain in a residence.

The normalization and deinstitutionalization of the mentally retarded population, a similar chronic population, who in some cases have similar behavioral manifestations, can be used as a model for providing this daily living assistance for those with AD in community residences supported by family or friend caretakers. The mentally retarded programs recognize an array of in home support services which enable individuals to live in a family or home setting, with case managers adapting the services to the specialized needs of the individual. These services might include home modifications such as the following: bathrooms with shower chairs and shower hoses to assist in bathing, doors that

are wheelchair accessible, alarm systems to alert if a person wanders through a door, stoves that will not light without special devices, special coded locks so that an individual cannot wander, alerts to notify police and fire departments that a challenged person is in the home, and labeling all items in the home so that a person with deteriorating cognitions can find needed items.

Services which are currently provided to individuals with behavioral conditions similar to AD can also be modified to include those with AD. For example, supportive day treatment services are available for the mentally challenged. These services could also provide a place where the individual with AD can safely stay during working hours. Since many with AD wander or do not sleep at night, these programs could follow the model of day care programs for children who assist shift workers and also offer the option of night care so that the caretaker can sleep. Nutritional services offered by a registered dietitian could help address, for example, the person with AD who goes through the agitated phase with the accompanying complication of weight loss. As the person loses eating skills an Occupational Therapist could assist in training dealing with how to modify food texture and assist in eating so the individual does not choke. Most importantly is the availability of respite care so that the caregiver can go to normal recreation, leisure, work and family activities, preventing caretaker burnout and stress. As cognitive functions decline, the person with AD needs a caretaker trained to help him or her eat, bathe, dress, toilet, brush teeth and remain occupied in daily leisure activities. Although at the latest stages, nursing home placement may be needed; there are stages during which care can be provided in a home or family setting with assistance from spouses, children or other family and friends.

Of the programs outlined above, the most widespread currently existing programs are support groups, most through the non-government voluntary system of the Alzheimer's Association. These support groups provide caretakers and family members with emotional support and practical techniques for dealing with an individual with AD. These groups also serve as a network for sharing information about improvements in services for the AD population quickly and easily transmitting information directly to the caregivers.

Finally nursing home care is usually the last choice at the end of the decision tree when family members and other caretakers are no longer able to manage the person with AD in their homes. The greatest portion of expense in nursing home care is in staff costs, which are anticipated to rise as the supply of workers lessens and their demand increases. The care for patients is labor intensive; as an individual with AD may present multiple symptoms and have co-morbidity.

### **Prevent or Delay the Onset of AD**

The second way of managing the expenses of AD is to delay the onset of the dementing processes. There are two possible ways of doing this, through pharmacological research, and the presence of protective lifestyles. Current medications only work to allay symptoms temporarily, if taken in the early phases of the disease. Brookmeyer and colleagues (2002) have noted that the current goal of pharmacological research is to allay symptoms for five years, significantly reducing the morbidity by AD. The cost of prescriptive medications will also need to be addressed.

Currently the U. S. has initiated the Medicare Part D program. In this system the consumer must make a computerized selection of private providers, from a range of

providers, each with a drug formulary. The person who selects the program does so for a year, limiting changing to another provider if newly prescribed drugs are not a part of the selected drug formulary. This program will be difficult for a demented person to access unless he or she has an advocate making the selection. Although this program does appear to privatize prescriptive medications one may question what will be the long term costs of this program as the population ages and requires multiple medications. One could question whether instead of waiting for private for profit pharmaceutical companies to invest in drug research and then sell the drugs at high cost, there should be government or voluntary non-government organization sponsored research to develop drugs to delay the onset of AD that could be mass produced, and sold as a generic drugs more cheaply to the growing aging population.

### **Lifestyle Issues**

Rogers and Hackenberg (1987) have suggested that the United States is in the hybristic stage of the Epidemiological transition with chronic and degenerative diseases influenced by personal choices. They noted that diet, exercise, refraining from smoking and moderate alcohol consumption are shown to increase both life span and decrease disability, thus lowering mortality by heart attacks, strokes, cancer, hypertension and diabetes. The U.S. population is now beginning to reap the benefits of a healthier lifestyle with a decrease in smoking lowering the rates of cancer. Researchers (Antuono and Beyer 1999, Miech and colleagues 2002, and Tschanz and colleagues 2004) have suggested that lifestyle choices including such personal choices of healthy life factors as education, nutrition and exercise are protective of AD. The literature also suggests that education and engaging in mentally stimulating activities are ways of delaying the onset

of AD. Researchers (Gatz 2000 Scarmeas et al. 2001, Stern et al 1994) found that mentally challenging leisure activities appeared to decrease the risk of AD. Since healthier lifestyles decrease overall mortality, AD researchers have suggested that both physical and mental exercise can decrease mortality or morbidity by AD.

This dissertation did not find that higher education levels were protective against mortality by AD. But discussion did address whether engaging in mentally stimulating activities would be protective of AD. More research is needed to determine if educational attainment and/or engaging in mentally or physically challenging activities protects one from AD. However studies, such as Bennett's (2004) longitudinal study of Religious Orders showed that mentally challenging activities seem to provide a reserve that protects against mental decline. Therefore policies, such as tuition free classes for elders at community and state colleges could be developed and expanded. Mentally challenging activities such as reading books, working crossword puzzles and playing math games along with physical exercise and senior Olympic Games should also be encouraged as protective of AD as well as the host of other chronic diseases. Finally the pathway from obesity is related to increased incidence of AD following vascular incidents, so obesity prevention may also be protective of AD.

### **Future Research**

In retrospect, this study could have benefited from additional analyses of the data. First there are four additional areas of study which could be undertaken with the NCHS multiple causes of death file data using Hierarchical Linear Modeling. Second, another area of study could combine NCHS survey data with existing mortality data. Third, international comparisons of incidence of AD could compare mortality by AD in



countries at different stages of the Epidemiological Transition. International comparisons could also address majority minority incidence of mortality by AD.

### *Hierarchical Linear Modeling*

First, one could expand on the analysis of existing data through statistical analysis. This study briefly noted the differences in ethnicity between the five Southwest states and Florida. Additional information could be obtained from the mortality data by an analysis using Hierarchical Linear Modeling (HLM) showing how mortality varies not only individually but also by state and by region of the country. The National Center for Health Statistics has analyzed the likelihood of mortality by state of 18 diseases, mapping their incidence by state; however AD is not one of the diseases they selected. First, states have different population age structures, ethnic concentrations, income rankings, and educational levels, as well as different portions of the population over 65. All of these factors could be addressed with their relationship to the likelihood of dying of AD. For example one HLM analysis could address mortality by AD, considering the age ranking of the population in the state.

Second, individuals who live in states may have different health patterns. The literature review noted for example that Cache County Utah has the longest lived persons. Rogers and colleagues (2000) note that Utah, with a largely Mormon population, follows a temperate lifestyle with limited alcohol and smoking, and has the longest life expectancy. Conversely Nevada, with a large tourism industry based on an excessive lifestyle, including ingestion of alcohol, has the shortest life expectancy. These two states have vastly different age structures as well as different life expectations due to lifestyle

choices. A model could then by proxy address lifestyle issues by comparing Mortality by AD in these two states.

Third, another issue which could be addressed through HLM is the ranking of states according to pollution indices. There is a literature that addresses the health hazards related to toxins. One of the areas of further research is to learn if there is an increase in mortality by AD in areas with high toxin levels.

Fourth, HLM by individual and state levels could address the internal migration issues. The migration of retirees is increasing. The healthy and wealthy are most likely to move to retirement communities, created and maintained to provide services to an aging society. It would be interesting to use rankings of desirability as a retirement location as a factor in an HLM analysis.

#### *NCHS Survey Data*

Another limitation of this research is that mortality alone does not completely address the issues of morbidity of AD, length of morbidity or the extent of services required. Since persons with AD live an average of between 5 and 20 years following diagnosis (Brookmeyer et al. 1998.), morbidity information is necessary for policy decisions based on projections for needed services for this increasing population. Brookmeyer and colleagues (2002) also found that length of survival after diagnosis depends on the age at onset, or original diagnosis, which would support a rectangularization theory. Those who have AD diagnosed in earlier ages survive with a morbidity of 7 to 10 years compared to the average length of survival being 3 years or less if diagnosed when in the 90's.

Morbidity issues could be addressed by including survey data presenting the care required by persons with AD compared to other chronic and degenerative diseases. There are three NCHS surveys which could be incorporated into an analysis of the morbidity of AD: the National Hospital Survey, the National Hospice and Home Health Care survey and the National Nursing Home Survey. These surveys provide data that address the duration and type of care received and the extent of mortality in AD care compared to those with other diagnoses. These data are necessary for financial projections about the future expenses in caring for an aging population with AD.

This analysis could include a description of the incidence of AD for males and females in age groups. Other descriptive data are also available through these surveys including diagnoses, reasons for admission and discharge from care, length of care and status of discharge. A hazard model analyses of the likelihood of dying of AD as compared to other causes of mortality analyzed during the duration of stay could address length of stay, discharge outcome, as well as types of care necessary upon discharge. These data could then be used with the information about the numbers of decedents with AD to more completely understand the extent of care and types of services a person with AD requires.

### *International Comparisons*

The world health organization encourages the use of the international classification of disease nomenclature, so international mortality due to AD comparisons can be based on the same coding system. International comparisons could show how the likelihood of cause specific mortality, including mortality by AD, is related to stages in the Epidemiological Transition. AD is one of the chronic and degenerative diseases

which are age related. It would be especially interesting to learn of mortality differences between developing and mature countries.

Another form of international analysis would be to address majority/minority status on an international basis. The analysis of data in this dissertation showed significant racial disparity in mortality by AD with Whites more likely to die of AD than ethnic minorities or non Hispanic Blacks. Data showed that NonHispanic blacks were less likely to die of AD than all other ethnic groups. One theory that supports the Black-White Mortality Crossover is that only the healthiest and hardiest of a minority population survives. Therefore they would be less susceptible to chronic and degenerative diseases such as AD. This raises the question of whether, using international comparisons, those minorities who survive to the oldest ages are hardier and therefore have a lower likelihood of mortality by AD. International comparisons, allowing one to address whether ethnicity or minority status is related to diagnosis of AD would allow for a more complete understanding of AD by ethnicity and majority status in the oldest old.

### **Conclusion**

This dissertation has raised the question of whether mortality by AD is inevitable with the global aging of the population. As the population ages there will be an increased likelihood of mortality due to chronic and degenerative diseases such as AD. The Center for Disease Control (CDC) estimates that in the United States the incidence of AD doubles every 5 years after age 65 with approximately 10 percent of adults  $\geq 65$  years, and 47 percent of adults  $\geq 85$  diagnosed with AD (CDC: 2003, 104). The research here found similar findings in the population of decedents from 1998-2002, with 4,619 dying of AD 116,444 aged 80 to 84; 127,113 aged 85 to 89. However, at age 90 to 94, the

numbers began to decline with 85,537 dying of AD as any condition. This decrease continued as 31,301 aged 95 to 99 decreasing to 4,923 for age 100 to 104 and 405 aged 105+. There is a different mortality pattern of AD among the oldest old. Mortality by AD peaks for decedents aged 85 to 89 and actually begins to decline for decedents over age 90. With the exceptions of marital status and education the hypotheses were supported. Females are more likely to die of AD than males. NonHispanic Whites are more likely to die of AD than Hispanics and NonHispanic Blacks. There is an increased risk of dying in a nursing home if one dies of AD. Future research as outlined above is needed to learn further about this fifth leading cause of mortality of those over age 60.

Four systems will need to respond: the health care and hospice system, the hospital system, the prescriptive drug system, and the long term care system. The current system of health in the United States is based on a fee for service acute care DRG model. Chronic and degenerative diseases require a system that is based on preventative care, stressing nutrition and healthy habits such as exercise and moderation. The health care system to meet the needs of an aging population must be able to deal with chronic conditions with alternatives for pain management and preventative health measures such as diet, exercise and avoidance of excesses. The countries of the world with the greatest longevity have medical care for all citizens, resulting in lower infant mortality rates and greater longevity. The United States does not.

As the population ages the concern is whether with increased age one will have increased disability. One of the gravest disabilities is AD, due to neurological degeneration and a lengthy progression. The positive findings of this study are that although AD is the fifth leading cause of mortality for those over age 60, the total

mortality due to AD, the definition being AD as underlying cause only, accounts for only 5 percent of mortality for those over 60.

## REFERENCES

- Abriaído-Lanza, A.F., Dohrenwend, B. P. & Ng-Mak D. S. et al. (1999) The Latino Mortality paradox; a test of the 'Salmon Bias' and Healthy Migrant Hypotheses, *American Journal of Public Health* 89: 1543-1548.
- Ahlburg, D. & Vaupel, J. W. (1990) Alternative Projection of the U.S. Population, *Demography*. 27:4, 639-652.
- Allison, P. D. (1984) *Event History Analysis: Regression for Longitudinal Event Data. Series: Quantitative Applications in the Social Sciences*, Newbury Park, California: Sage Publications.
- American Psychiatric Association. (1994) *Diagnostic and Statistical Manual of Mental Disorders 4<sup>th</sup> Ed.* Washington D.C.: APA.
- Anderson, R. N., Minino A. M., Hoyert D. & Rosenberg H. (2001) Comparability of Cause of Death Between ICD-9 and ICD-10: Preliminary Estimates, *National Vital Statistics Reports*. 49:2, 1-32.
- Antuono, P. & Beyer J. (1999) The Burden of Dementia: A Medical and Research Perspective, *Theoretical Medicine and Bioethics* 20:1, 3-13
- Azar, B. (2002) Use it or Lose it, *American Psychological Association Monitor site* Accessed at: <http://www.apa.org/monitor> May 2004.
- Barnes, L. L, Wilson, R. S., Bienias, J. L., Schneide, J. A., Evans, D. A. & Bennett D. A. (2005) Sex Differences in the Clinical Manifestations of Alzheimer Disease Pathology, *Archives General Psychiatry*. 62: 685-691.
- Bassuk, S. S. & Glass, T. A.(1999) Social Disengagement and Incident Cognitive Decline in Community-dwelling Elderly Persons, *Annals of Internal Medicine* 131: 165-174.
- Bennett, D. A. MD (2004) Take-home Lesson: Formal Education May Help Protect the Brain Against Alzheimer's Disease, *Neurology* June 24, 2004.
- Berkman, L. F. (2000) Which Influences Cognitive Function: Living Alone or Being Alone? *Lancet* 355:1291-2.
- Berkman, L., Singer, B. & Manton K. Black/White Differences in Health Status and Mortality among the Elderly, *Demography* 26(4) 61-678.
- Blieszner, R. & Alley, J. M. (1990) Family Caregiving for the Elderly: An Overview of Resources, *Family Relations* 39:97-102.

- Blieszner, R. & Shifflett, P. (1990) The Effects of Alzheimer's Disease on Close Relationships Between Patients and Caregivers, *Family Relations* 39: 57-62.
- Bonsignore, M., Barkow, K. & Heun, R. (2002) Possible influence of selection bias on Gender Differences in the Risk of Alzheimer's Disease. *Archives of Women's Mental Health* 5:2, 73-77.
- Brookmeyer, R., Gray, S. & Kawas, C. (1998) Projections of AD Disease in the United States and the Public Health Impact of Delaying Disease Onset, *American Journal of Public Health* 88:9, 1337-1342.
- Brookmeyer, R., Corrada, M., Curriero, F. & Kawas, C. (2002) Survival Following a Diagnosis of Alzheimer's Disease, *Archives of Neurology* 59:1764-1767.
- Burns, A., Jacoby, R., Luthert, P. & Levy, R. (1990) Cause of Death in Alzheimer's Disease, *Age and Ageing* 19, 341-344.
- Caldwell, John C. (2001) Demographers and the Study of Mortality: Scope, Perspectives and Theory, *Epidemiology and Demography* 954:175-183.
- Carey, J. R. (2003) *Longevity: The Biology and Demography of the Life Span*, Princeton, New Jersey and Oxford: Princeton University Press.
- Carnes, B. A. & Olshansky, S. J. (1993) Evolutionary Perspectives on Human Senescence, *Population and Development Review*. 19:4, 793-806.
- Center for Disease Control (2004) Health Disparities Experienced by Hispanics—United States, *Morbidity and Mortality Weekly Report* 53: 935-937.
- Center for Disease Control (1990, 1) Mortality from Alzheimer's Disease—United States, 1979-1987, *Morbidity and Mortality Weekly Report* 39: 785-788.
- Center for Disease Control (1990, 2) Progress in Chronic Disease Prevention Alzheimer's disease—California, *Morbidity and Mortality Weekly Report* 39:105-107.
- Center for Disease Control (2003) Trends in Aging—United States and Worldwide, *Morbidity and Mortality Weekly Report* 52: 101-106.
- Dalsania, P. (2004) Dementias Other than Alzheimer's In: K. J. Doka (ed.), *Living with Grief: AD*. Washington, D.C.: Hospice Foundation of America.
- Davis, M. A., Poston, D. L., Jr., & Min. H. (2004) A Comparison of Health



- Conditions in the Deaths from Alzheimer's Disease in the Elderly between South Korea and the United States. *The Journal of Gerontology* 8: 203-224.
- Deaths, percent of total deaths, and death rates for the 15 leading causes of death in 5-year age groups, aged 60-100+ all races and both sexes: United States, 2002  
Accessed at: <http://www.cdc.gov/nchs/data> May 2005.
- Dobalian, A., Tsao, J.C., & Radcliff, T.A. (2003). Diagnosed mental and physical Health conditions in the United States Nursing Home Population: Differences between Urban and Rural Facilities, *Journal of Rural Health* 19:4 477-483.
- Dodge, H. H., Shen, C., Pandav, R., DeKosky, S. T. & Ganguli, M. (2003) Functional Transitions and Active Life Expectancy Associated with Alzheimer's Disease *Archives of Neurology* 60: 253-259.
- Elman, C. & Uhlenberg, P. (1995) Co-residence in the Early Twentieth Century: Elderly Women in the United States and Their Children . *Population Studies*. 49(3) 501-517.
- Elo, I. T. & Preston, S. H.. (1992) Effects of Early-Life Conditions on Adult Mortality: A Review, *Population Index* 58 (2): 186-212.
- Franzini, L., Ribble, J.D. & Keddie, A. M (2001) Understanding the Hispanic Paradox, *Ethnicity and Disease*.11: 496-518
- Fratiglioni L; Paillard-Borg, S. & Winblad, B. (2004) An Active and Socially Integrated Lifestyle in Late Life Might Protect Against Dementia, *Lancet* 3: 343-53.
- Fratiglioni, L., H., Wang X., Ericsson, K., Maytan & Winblad, M. B. (2000) Influence of Social Network on Occurrence of Dementia: A Community-based Longitudinal Study, *Lancet* 355: 1315-9.
- Freeman, V.A. (1993) Kin and Nursing Home Lengths of Stay: A Backward Recurrence Time Approach, *Journal of Health and Social Behavior* 34 (2) 138-152.
- Freeman, V. A. & Martin, L. G. (1999). The Role of Education in Explaining and Forecasting Trends in Functional Limitations among Older Americans, *Demography* 36:461-473.
- Fuh, J. L. (2003) Patients with Dementia and Their Caregivers in Taiwan in Eileen M. Welsh Ed. *Focus on Alzheimer's Disease Research* . New York: Nova Biomedical Books.
- Gambassi, G., Landi, F., Lapane, K. L., Sgadari, A., Mor, V.

- & Bernabei, R. (1999) Predictors of Mortality in Patients with Alzheimer's Disease Living in Nursing Homes, *Journal of Neurology Neurosurgery Psychiatry* 67:59-65.
- Gandy, S., Martins, R. N. & Buxbaum. J. (2004) AD: Biology and Therapy in Doka, K. J. Ed. *Living with Grief: AD Disease*. Washington, D.C.: Hospice Foundation of America.
- Ganguli, M., Dodge, Hiroko H., Shen, C., Pandav, R. S., DeKosky S. T. (2005) Alzheimer Disease and Mortality: A 15 –Year Epidemiological Study, *Archives of Neurology* 62:779-784.
- Gao, S. Hendrie, H., Hall, K.S., and Hui A. The Relationships Between Age, Sex, and the Incidence of Dementia and Alzheimer Disease: Meta-analysis *Archives General Psychiatry*. 1998; 55:809-815.
- Gatz ,M., Fiske, A. , Reynolds, C. A. Loebach Wetherell, J. , Johansson, B. & Pedersen, N. L. (2003) Sex Differences in Genetic Risk for Dementia Behavior, *Genetics* 33:2, 95 – 105.
- Gatz M., Pedersen, N.L., Crowe, M., Fiske, A. (2000) Defining Discordance in Twin Studies Of Risk and Protective Factors for Late Life Disorders. *Twin Research* 3 (3):159-64.
- Geronimus, A. T., Bound, J., Waidmann, T. A., Coen, C. G., Steffick, D. (2001) Inequality in Life Expectancy, Functional Status and Active Life Expectancy across Selected Black and White Populations in the United States, *Demography* 38 (2): 227-251.
- Goldman, N. (2001) Social Inequalities in Health: Disentangling the Underlying Mechanisms, *Annals of the New York Academy of Sciences* 954:118-139.
- Green R. C., Adrienne, L. Cupples, R., Kelly G., Benke, S., Edeki, T., MD, Griffith, P., Williams, M. A., Hipps, Y., Graff-Radford, N., Bachman, D. Farrer, L. A., (2002) Risk of Dementia Among White and African American Relatives of Patients With Alzheimer Disease . *JAMA*: 287:329-336.
- Hamilton, L. C. (1992) *Regression with Graphics: A Second Course in Applied Statistics*. Duxbury Press: Belmont, California.
- Harwood, D. G., Ownby, R. L. , Burnett, K., Barker, W. W. & Duara.R. (2000) Predictors of Appraisal and Psychological Well-Being in Alzheimer's Disease Family Caregivers, *Journal of Clinical Geropsychology* 6:4, 279-297.

- Hay, J. W. & Ernst, R. L. (1987) The Economic Costs of AD, *American Journal of Public Health* 77:1169-1175.
- Hay, J. W. & Ernst, R. L. (1994) The U. S. Economic and Social Costs of AD Revisited, *American Journal of Public Health* 84:1262-1264.
- Hebert, L. E., Scherr, P. A., Bienias, J. L., Bennett, D. A. & Evans, D. (2003) Alzheimer Disease in the U. S. Population: Prevalence Estimates using the 2000 Census, *Archives of Neurology* 60:8, 1119-1122.
- Helmer, C., Joly, P., Letenneur, L., Commenges, D. & Dartigues, J - F. (2000) Mortality with Dementia: Results from a French prospective Community Based Cohort *American Journal of Epidemiology*.154:642-648.
- Heininger, K. (2000) A Unifying Hypothesis of Alzheimer's Disease. III. Risk Factors *Human Psychopharmacology Clinical Experience*. 15: 1-70.
- Hendrie H. C., Ogunniyi, A., Hall, K. S., Baiyewu, O.; Unverzagt F. W., Gureje. O., Gao, S., Evans, R. M.; Ogunseyinde, A. O., Adeyinka A. O.; Musick, B. Hui S. L. (2001) Incidence of Dementia and Alzheimer Disease in 2 Communities: Yoruba Residing in Ibadan, Nigeria, and African Americans Residing in Indianapolis, Indiana *JAMA*, 285: 739 - 747.
- Hetzel, A. M. (1997) History and Organization of the Vital Statistics System. National Center for Health Statistics: Hayattsville, Maryland.
- Heun R. & Kockler, M. (2002) Gender differences in the cognitive impairment in Alzheimer's disease, Accessed at: <http://alzfdn.org/alzheimers/index.shtml> May 2005.
- Hofman, W. A., Rocca, Brayne C, Breteler, M. Clarke, M., Cooper, B., Copeland, J. R. M., Dartigues, J. F., Da Silva Droux, A., Hagnell, O., Heeren, T. J., Engedal, K., Jonker, C., Lindesay J, Lobo, A., Mann, A. H., Mölsäm P, K., Morgan, K., O'Connor, D. W., Sulkava, R., Kay, D. W. K. & Amaducci L., (1991) The Prevalence of Dementia in Europe: A Collaborative Study of 1980–1990 Findings, *International Journal of Epidemiology* 20: 736-748.
- Holstein, M. (2000) Aging Culture and the Framing of Alzheimer Disease in Concepts of Alzheimer Disease: Biological, Clinical and Cultural Perspectives (eds.), Whitehouse, Konrad, P. J., Maurer & Ballenger, J. F.. Baltimore: The John Hopkins University Press.
- Horiuchi, S. & Wilmoth, J. R (1998) Deceleration in the Age Pattern of Mortality at Older Ages, *Demography*. 35; 391-412.

- Hoyert, D. L. (1996) Mortality Trends for AD, 1979-91. National Center for Health Statistics, Vital Health Stat 20 (28).
- Hoyert, D. L. (2001) The Autopsy, Medicine and Mortality Statistics. National Center for Health Statistics: Hayattsville, Maryland.
- Hoyert, D. L., Arias, E., Smith, B.L., Murphy, S. L. & Kochanek, K. D. (2001) Deaths: Final Data for 1999 (Technical Notes and References . National Vital Statistics Reports 49: 90-115.
- International Classification of Diseases, 9th Revision, Clinical Modification. (ICD 9 CM) Accessed at: <http://www.cdc.gov/nchs/datawh/ftpserve/ftp9icd9/ftp9icd9.htm>. May 2005.
- Johnson, N. E. (2000) "The Racial Crossover in Comorbidity, Disability and Mortality". (2000), Demography 37: 267-283.
- Katzman, R. & Bick, K. (2000) Alzheimer Disease: The Changing View. New York: Academic Press.
- Kimberly Mooney Munez Admission Severity and Mortality Rates Among Rural And Urban Facility Rates with Dementia March 2001, Working Paper. Accessed at: <http://www.alz.org/Media/newsreleases> May 2005.
- Kochanek, K.D., Murphy, S. L., Anderson, R. & Scott, C. (2004) Death Final Data For 2002, National Vital Statistics Reports 53: 1-116.
- Koester, R. J. The Lost Alzheimer's and Related Disorders Search Subject: New Research & Perspectives Accessed At: [http://www.dbs-sar.com/SAR\\_Research/lost\\_alzheimer.htm](http://www.dbs-sar.com/SAR_Research/lost_alzheimer.htm). May 2005.
- Lapane, K. L., Gambassi, G., Landi, F.F., Sgadari, A., Mor, V. & Bernabei, R. (2001) Gender differences in predictors of mortality in nursing home residents with AD, Neurology 56: 650-654.
- Larson, E. B., Shadlen, M.-F., Wang, L., McCormick, W. James, C., Bowen, D., Teri, L. & Kukull, W. A. (2004) Survival after Initial Diagnosis of Alzheimer's Disease Annals of Internal Medicine. 140:501-509.
- Long, J. S. & Freese, J. (2003) Regression Models for Categorical Dependent Variables Using Stata. Stata Press: College Station, Texas.
- Long, J. S. & Freese, J. (2006) Regression Models for Categorical Dependent Variables Using Stata. Second Edition. Stata Press: College Station, Texas.
- Lynch, S. M, Brown J. S. & Harmsen, K. G. (2003) Black-white

Differences in Mortality Compression and Deceleration and the Mortality Crossover Reconsidered. *Research on Aging* 25: 456-483.

- McKibben, S. L. (2005) The Social Construction of Adulthood: Menarche and Motherhood. Texas A&M Dissertation. Accessed at: <http://handle.tamu.edu/1969.1/1645>. May 2005.
- McMurty, S. L. & Gwat-Yong Lie Differential Exit Rates of Minority Children in Foster Care. *Social Work Research and Abstracts*: 28 (1) 42-55.
- Magnetic Resonance Imaging (MRI) of Brain Atrophy in Alzheimer's Disease, Accessed At: <http://www.mayoclinic.com>. May 2005.
- Manton K. G., Stallard E. & Tolley, H. D. (1991) Limits to Human Life Expectancy: Evidence, Prospects, and Implications .*Population and Development Review*. 17:4,603-637.
- Manton, K. G. & Gu, X (2001) Changes in the Prevalence of Chronic Disability in the United States Black and Non-black Population above age 65 from 1982-1999 . *Proceedings of the National Academy of Sciences of the United States of America*. 98; 6354-59.
- Mathers, C. D., Sadana, R., Salomon, J. A., Murray, C. J. L. & Lopez, A.(2000) Estimates of DALE for 191 Countries: Methods and Results . *Global Programme on Evidence for Health Policy*. Working Paper Accessed at: <http://www.who.int/health-systems-performance>. May 2005.
- McFalls, J.(2003) Population: A Lively Introduction. *Population Bulletin*, December. Accessed at: <http://www.prb.org/pdf/populationlivelyintro> January 2005.
- Meuser, T. M, Marwit, S. J. & Sanders, S. (2004) Assessing Grief in Family Caregivers in Doka, K. J. (ed.), *Living with Grief: AD Disease*, Washington, D.C.: Hospice Foundation of America.
- Miech, R. A., Breitner, J. C. S., Zandi, P., Kachaturian, A. S., Anthony, J. C., & Mayer, L. (2002) Incidence of AD May Decline in the Early 90's for Men, Later for Women. *The Cache County Study Neurology*.58: 209-218.
- Mitchell, S. L., Kiely, D. K., Hamel, M. B., Park, P. S., Morris, J. N. & Fries, B. E. (2004) Estimating Prognosis for Nursing Home Residents with Advanced Dementia, *Journal of the American Medical Association* 291:2734-2740.
- Mortality Data, Multiple Cause-of-Death Public-Use Data Files Data File Description: Detail (1999-2002) Accessed at:

[http://www.cdc.gov/nchs/products/elec\\_prods/subject/mortmcd.htm](http://www.cdc.gov/nchs/products/elec_prods/subject/mortmcd.htm). January 2005.

- Myers, L. J., Stokes, DeV. R. & Speight, S. L. (1989) Physiological Responses to Anxiety and Stress: Reactions to Oppression, Galvanic Skin Potential, and Heart Rate, *Journal of Black Studies* 20 (1) 80-96.
- Nam, C. B. (1995) Another Look at Mortality Crossovers. *Social Biology* 42: 133-142.
- National Center for Health Statistics (1998) A Guide to State Implementation of ICD-10 for Mortality. Accessed at: [http://ftp.cdc.gov/pub/Health\\_Statistics/NCHS/Publications/ICD9\\_10](http://ftp.cdc.gov/pub/Health_Statistics/NCHS/Publications/ICD9_10) May 2005.
- National Center for Health Statistics. (2004) 1998 Multiple-Cause-of-Death File, NCHS CD-ROM. Unpublished. Hyattsville, Maryland: National Center for Health Statistics.
- National Center for Health Statistics. (2004) 1999 Multiple-Cause-of-Death File, NCHS CD-ROM. Unpublished. Hyattsville, Maryland: National Center for Health Statistics.
- National Center for Health Statistics. (2004) 2000 Multiple-Cause-of-Death File, NCHS CD-ROM. Unpublished. Hyattsville, Maryland: National Center for Health Statistics.
- National Center for Health Statistics. (2004) 2001 Multiple-Cause-of-Death File, NCHS CD-ROM. Unpublished. Hyattsville, Maryland: National Center for Health Statistics.
- National Center for Health Statistics. (2004) 2002 Multiple-Cause-of-Death File, NCHS CD-ROM. Unpublished. Hyattsville, Maryland: National Center for Health Statistics.
- National Center for Health Statistics. (1998) A Guide to State Implementation of ICD-10. Accessed at: <http://www.openclinical.org/medTermICD>. May 2005.
- New Alzheimer's Association Report Predicts Disease Will Soar 600 Percent Among Hispanics by 2050 Accessed at: [http://www.alz.org/Media/newsreleases/2004/040511\\_eng.asp](http://www.alz.org/Media/newsreleases/2004/040511_eng.asp). May 2005.
- Nocera, S., Telser, H. & Bonato, D. (2003) *The Contingent Valuation Method in Health Care: An Economic Evaluation of AD*. Boston: Kluwer Academic Publishers.
- Olshansky, S. J. (2003) From Michelangelo to Darwin: the Evolution of Human Longevity . *IMAJ* 5: 1-3.

- Olshansky, S. J. & Ault, B. (1986) The Fourth Stage of Epidemiologic Transition: the Age of Delayed Degenerative Diseases, *The Milbank Quarterly*. 64:3 355-391.
- Olshansky S. J.; Carnes, B. A.. (1994) Demographic Perspectives on Human Senescence . *Population: An Introduction to Concepts and Issues* Ninth Edition. Development Review, 20:1, 57-80.
- Olshansky S. J.; Carnes, B.A. & Desesquelles, A. (2001) Demography: Prospects for Human Longevity, *Science*, 291, 1491-1492.
- Olshansky S. J.; Carnes, B. A.; Cassel, C.. (1990) In Search of Methuselah: Estimating the Upper Limits to Human Longevity *Science*. 250:4981, 634-640.
- Olshansky S. J.; Hayflick, L. & Perls, T. T. (2004) Anti-Aging Medicine: the Hype and the Reality-part II . *Journal of Gerontology*. 59A:7 649-751.
- Omran, A. (1971) The Epidemiologic Transition: A Theory of the Epidemiology of Population Change . *The Milbank Quarterly*. 49: 509-538.
- Palloni, A. & Morenoff, J. D. (2001) Interpreting the Paradoxical in the Hispanic Paradox: Demographic and Epidemiological Approaches . *Annals of the New York Academy of Science*. 954: 140-74.
- Pollen, D, A. (2000) The History of the Genetics of Alzheimer Disease in Peter J. Whitehouse, Konrad Mauer & Jesse F. Ballenger Eds. *Concepts of Alzheimer Disease: Biological, Clinical, and Cultural Perspectives* Baltimore: Johns Hopkins University Press.
- Population Pyramid Summary for United States Accessed at:  
<http://www.census.gov/ipc/www/idbpyr.htmlprod/wp02/wp-02004>. May 2005.
- Poston, D. L. Jr. Unpublished notes for Sociology 631 Sociological Research: Quantitative Methods Lecture 7, Spring, 2003.
- Poston, D. L. Jr., Davis, M. A. & Lewinski, C. Mortality (2005 forthcoming) In the *Cambridge Dictionary of Sociology*. Bryan Turner Ed. Cambridge, England: Cambridge University Press.
- Poston, D., L. Jr. & Kim, H G. (1999) The Elderly Populations of South Korea and North Korea: Current and Projected Levels and their Implications . *Journal of the Korea Gerontological Society* 19(3) 187-197.
- Poston, D., L. Jr. & Min, H. (2003) Cause of Death of the Oldest Old in the

Republic of Korea in 2001 With Comparisons to the United States in 1998.  
Unpublished document.

Preston, S. H., Elo, I. T., Rosenwaike, I. & Hill, M. (1996) African-American Mortality at Older Ages: Results of a Matching Study *Demography*. 33: 193-209.

Raftery, A. E. (1995) Bayesian Model Selection in Social Research (with Discussion) in *Sociological Methodology* 1995 P. V. Marsden ed. Cambridge, Mass.: Blackwell Publishers.

Rapp, M., Beeri, M., Schmeidler, J., Sano, M., Silverman, J. M., Haroutunia, V.(2005) Relationship of Neuropsychological Performance to Functional Status in Nursing Home Residents and Community-Dwelling Older Adults, *American Association for Geriatric Psychiatry*. 13:450-459.

Ritchie, K. & Kildea, D. (1995) Is senile dementia age-related or aging related –evidence from meta-analysis of dementia prevalence in the oldest old, *The Lancet* 346:931-934

Rogers, R. G., & Hackenberg, R. (1987) Extending Epidemiologic Transition Theory: A New Stage. *Social Biology* 34: 234-243.

Rogers, R. G., Hummer, R. A. & Krueger, P. M. (2005) Adult Mortality, in D. L. Poston, D. L., Jr. & Micklin, M. (eds.), *Handbook of Population*, New York: Springer.

Rogers, R. G., Hummer, R. A. & Nam, C. B. (2000) *Living and Dying in the USA: Behavioral, Health and Social Differentials of Adult Mortality*. San Diego, California.: Academic Press.

Rogers, R. G., Hummer, R. A. & Nam, C. B. & Peters, K.. (1996) Demographic, Socioeconomic and Behavioral Factors Affecting Ethnic Mortality by Cause *Social Forces* 74:4 1519-1438.

Rowland, D. T. (2003) *Demographic Methods and Concepts* New York: Oxford University Press.

Saenz, R. & Morales, M. C. (2005) Demography of Race and Ethnicity In *Handbook of Population*, edited by Poston, D. L., Jr. & Micklin, M.. New York: Springer.

Saenz, R. & Rubio, M. (2004) A Sociodemographic Profile of Elderly Mexicans in the United States: the Case of the 0.25 Generation and Beyond Unpublished Paper presented at the Colorado Conference on the Estimation of Migration. Estes Park, Colorado September 24-26, 2004.

Sahyoun, N. R. Lentzner, H., Hoyert, D. & Robinson, K.N. (2001) Trends In Causes of



Death Among the Elderly Aging Trends; No.1. Hyattsville, Maryland: National Center for Health Statistics.

Scarmeas, N. G., Levy, M., Tang S., Manly, J. & Stern Y. (2001) Influence of leisure activity on the incidence of Alzheimer's disease *Neurology*. 57: 2236-2242.

Schoenborn, C. A. (1986) Healthy Habits of U.S. Adults, 1985: the 'Alameda' Revisited. *Public Health Reports*. 101:6 571-580.

Schulz, R., et al. Resources for Enhancing Alzheimer's Caregiver Health, 1996-2001 [Computer file]. ICPSR version. Pittsburgh, PA: Richard Schultz, University of Pittsburgh [producer], 2001. Ann Arbor, MI: Inter-University Consortium for Political and Social Research [distributor], 2003.

Seeman, T. E. & Crimmins, E. (2001) Social Environmental Effects on Health and Aging: Integrating Epidemiological and Demographic Approaches and Perspectives, *Annals of the New York Academy of Sciences* 954:88-117.

Shakespeare, William As you Like It. Accessed at: <http://www-tech.mit.edu/Shakespeare> May 2005.

Shyrock, H. & Siegel, J. (1973) *The Methods and Materials of Demography*. Washington: US Bureau of the Census, US Government Printing Office

Stern, Y, Gurland, B, Tatemichi, T.K., Tang, M.X., Wilder, D., & Mayeux, R. (1994) Influence of education and occupation on the incidence of Alzheimer's disease *Journal of the American Medical Association* 271:1004-1010.

Tschanz, J. T., Corcoran, C., Skoog, I., Khachaturian, J. A., Herrick, S., Hayden, K. M., Welsh-Bohmer, K. A., Calvert, T., Norton, M. C., Zandi, P., & Breitner J. D. S. (2004) Dementia: The leading predictor of death in a defined elderly population: The Cache County Study. *Neurology*; 62: 1156-1162.

Uhlenberg, P. (1992) Population Aging and Social Policy *Annual Review of Sociology* 18: 449-474.

U.S. Census Bureau, International Data Base. Accessed at: <http://www.prb.org>. Population Reference Bureau January 2005.

U.S. Census Bureau, International Population Reports WP/02. (2004) *Global Population Profile: 2002* U. S. Government Printing Office: Washington D.C.

U.S. Department Of Health And Human Services: Instructions for Completing the Cause-of-Death Section of the Death Certificate, Accessed at: [http://www.cdc.gov/nchs/data/dvs/blue\\_form.pdf](http://www.cdc.gov/nchs/data/dvs/blue_form.pdf) May 2005.

- Vaupel, J. (2001). Demographic Insights into Longevity .Population: An English Selection, 13: 1, 245-259.
- Vaupel, J. (1997). The Remarkable Improvements in Survival at Older Ages Philosophical Transactions: Biological Sciences, 352:1363, 1799-1804.
- Vaupel, J. W., Carey, J. R., Christensen, K., Johnson, T., Anatoli E., Yashin, I. Holm, N. V., Iachine, I.A., Kannisto, V., Khazaeli, A. A., Liedo, P, Longo, V. D., Zeng, Y., Manton, K. G. & Curtsinger, J. W.(1998). Biodemographic Trajectories of Longevity . Science 280:5365, 855-860.
- Watari, Kecia F. & Gatz, M. (2004) Pathways to Care for Alzheimer's Disease Among Korean Americans . Cultural Diversity and Ethnic Minority Psychology. Vol. 10(1): 23-38.
- Watkins, S. C., Menken, J. A. & Bongaarts, J. (1987) Demographic Foundations of Family Change American Sociological Review. 52 (3) 346-358.
- Weeks, J. R. (2005) Population: An Introduction to Concepts and Issues Ninth Edition. Wadsworth: Belmont, California.
- Wilmo, A., Winblad, B., Aguero-Torres, H. & von Strauss E. (2003) The Magnitude of Dementia Occurrence in the World Alzheimer's Disease and Associated Disorders.17: 63-67.
- Wilmoth, J. R. & Horiuchi, S. (1999) Rectangularization Revisited: Variability of Age at Death Within Human Populations . Demography 36:475-495.
- Wolfson, C., Wolfson, D. Asgharian, B. M., M'Lan, C. E., Óstbye, T. Rockwood, K.& Hogan D. B (2001) The New England Journal of Medicine. 344:1111-1116.
- Yeo, G. & Gallagher, D. Ed. (1996) Burden of Mental and Behavioral Disorders . Ethnicity and the Dementias. Thompson. Washington, D.C.: Taylor & Francis.
- Zandi, P., Michelle P, Carlson,C., Plassman,B. L., Welsh-Bohmer K., Mayer, L. S., Steffens, D. C. & Breitner, J.C. S. (2002) Hormone Replacement Therapy and Incidence of Alzheimer Disease in Older Women: The Cache County Study Journal of the American Medical Association 288:2123-2129.

## APPENDIX A

**Table A.1: Midyear Population U. S. 2025 and 2050 by Age 60 + & Sex**

Country /Year	Population both	Population male	Population female	Percent total	Percent male	Percent female
		Sex ratio				
United States/2000		96.4		Median age 35.3		
60- 64	10,864,730	5,165,703	5,699,027	3.8	3.7	4
65- 69	9,533,955	4,402,844	5,131,111	3.4	3.2	3.6
70- 74	8,849,946	3,904,321	4,945,625	3.1	2.8	3.4
75- 79	7,425,378	3,051,227	4,374,151	2.6	2.2	3
80+	9,251,968	3,093,305	6,158,663	3.3	2.2	4.3
Total	282,338,631	138,595,702	143,742,929	100	100	100
		Sex ratio				
United States/2025		96.7		Median age 38.5		
60- 64	21,128,456	10,184,920	10,943,536	6	5.9	6.2
65- 69	19,646,750	9,283,604	10,363,146	5.6	5.4	5.8
70- 74	16,040,825	7,346,016	8,694,809	4.6	4.3	4.9
75- 79	12,267,624	5,376,751	6,890,873	3.5	3.1	3.9
80+	15,568,533	5,793,098	9,775,435	4.5	3.4	5.5
Total	349,666,199	171,918,242	177,747,957	100	100	100
		Sex ratio				
United States/2050		96.8		Median age 39.1		
60- 64	22,384,189	10,997,678	11,386,511	5.3	5.3	5.3
65- 69	20,443,823	9,911,357	10,532,466	4.9	4.8	4.9
70- 74	17,498,614	8,273,629	9,224,985	4.2	4	4.3
75- 79	15,066,841	6,853,651	8,213,190	3.6	3.3	3.8
80+	33,696,359	13,289,512	20,406,847	8	6.4	9.6
Total	420,080,587	206,672,139	213,408,448	100	100	100

Source: U.S. Bureau of the Census, International Data Base.

Table A.2: Projected Dependency Ratios for the U. S. 2000, 2025 and 2050

	2000	2025	2050
Dependency Ratio 0-14	32.26	32.12	32.93
Dependency Ratio 65+	18.75	29.33	34.57
Total Dependency Ratio	51.01	61.46	67.50
% over 65	12.4	18.2	14.7
Median Age	35.3	38.5	39.1
Sex Ratio	96.4	96.7	96.8

Source: U.S. Bureau of the Census International Data Base

Table A.3 Percentages of Mortality by AD by Age in the U. S. in 2001

Age	Males	Females
60's	.71	.04
70's	2.30	2.78
80's	4.48	5.52
90's	4.61	5.86
100's	3.21	4.07

Source: Davis et al. 2004

Figure A.1: Magnetic Resonance Imaging (MRI) of Brain Atrophy in Alzheimer's Disease



**Normal**



**Mild Alzheimer's**



**Moderate Alzheimer's**



**Severe Alzheimer's**

Figure A.2: Population Pyramid Summary for the United States

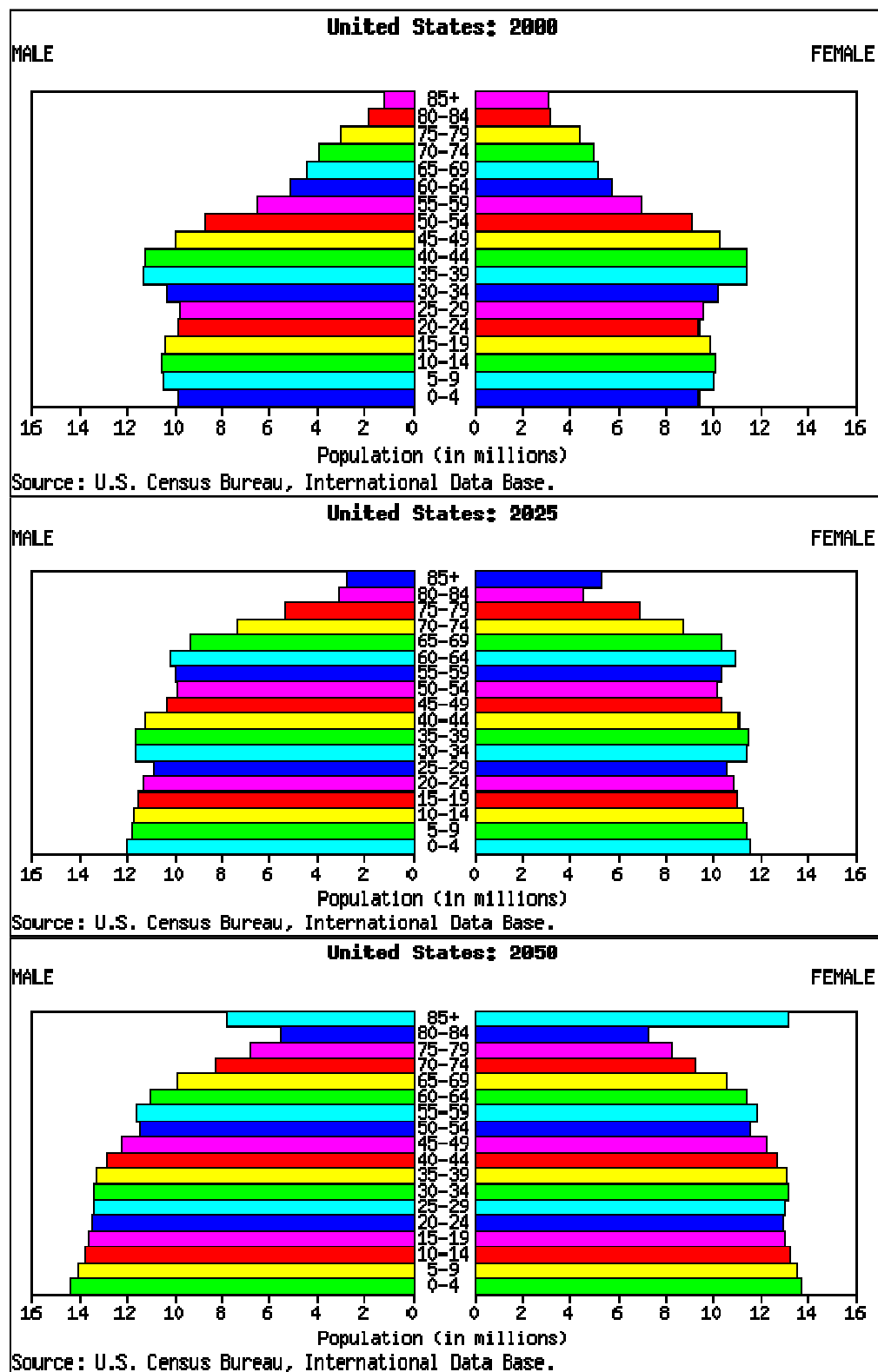


Figure A.3: Percentage of Mortality by Alzheimer's Disease 2001 by Age

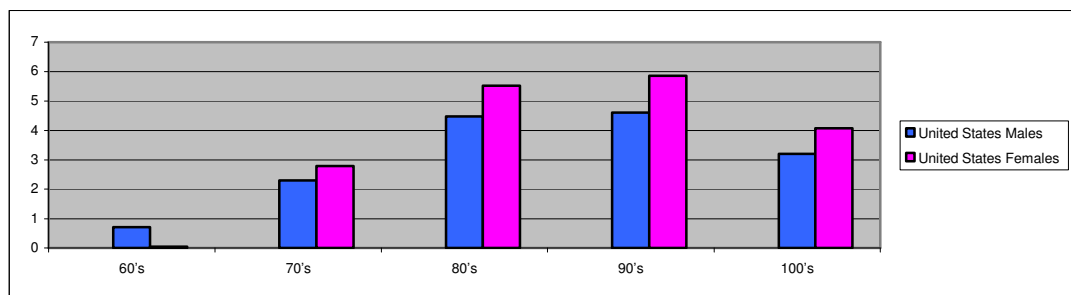


Figure A.4: Cause of Mortality among Decedents Age 60's in the U. S. 2001

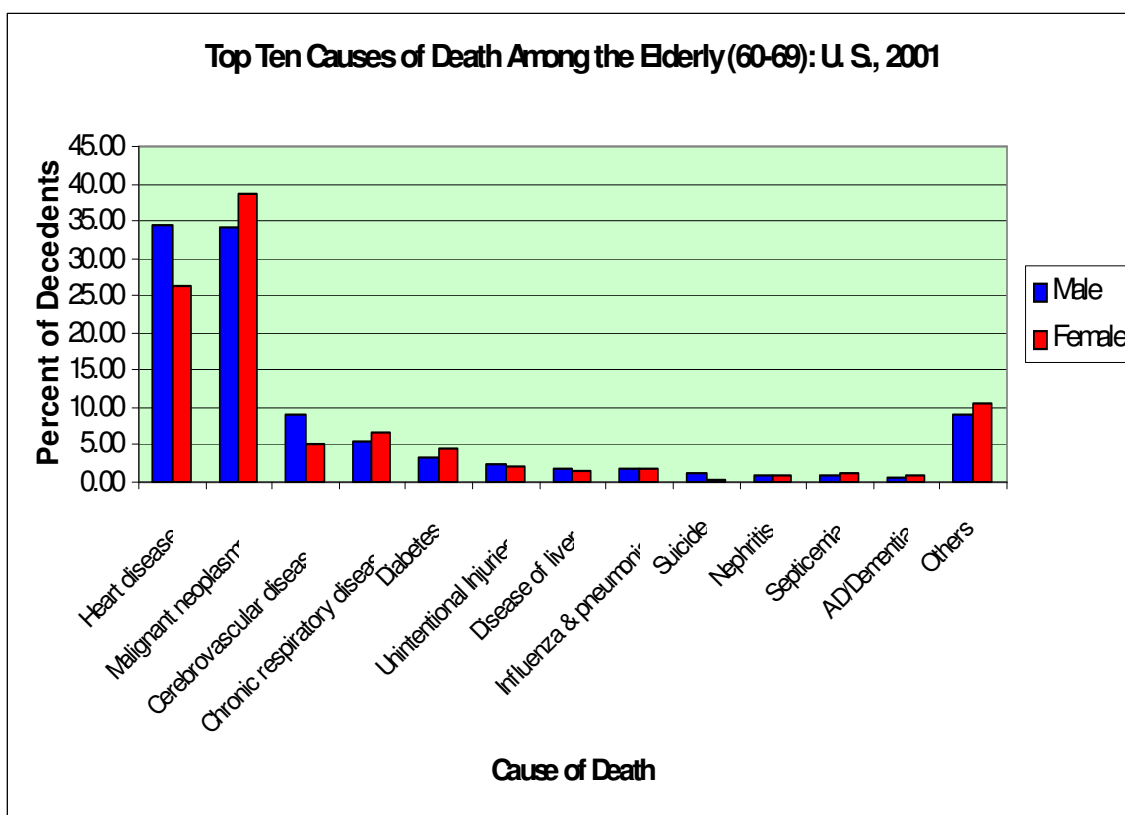
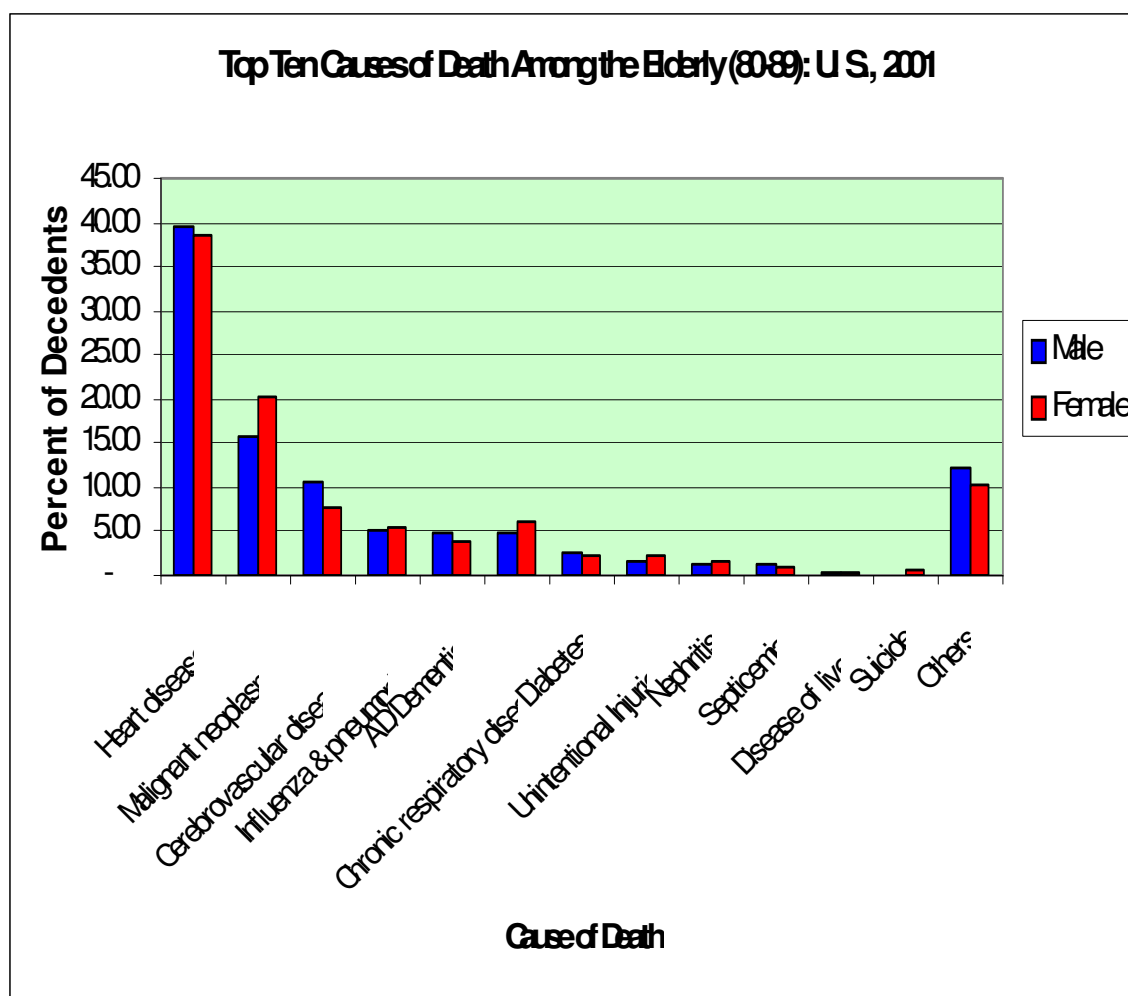


Figure A.5: Cause of Mortality among Decedents Age 80's in U. S.





## APPENDIX B

### Instructions for Completing the Cause-of-Death Section of the Death Certificate

Accurate cause-of-death information is important:

- To the public health community in evaluating and improving the health of all citizens, and
- Often to the family, now and in the future, and to the person settling the decedent's estate.

The cause-of-death section consists of two parts. **Part I** is for reporting a chain of events leading directly to death, with the **immediate cause** of death (the final disease, injury, or complication directly causing death) on Line a and the **underlying cause** of death (the disease or injury that initiated the chain of morbid events that led directly and inevitably to death) on the lowest used line. **Part II** is for reporting all other significant diseases, conditions, or injuries that contributed to death but which did not result in the underlying cause of death given in **Part I**. **The cause-of-death information should be YOUR best medical OPINION.** A condition can be listed as “probable” even if it has not been definitively diagnosed.

#### Examples of properly completed medical certifications

<b>32. PART I.</b> Enter the chain of events—diseases, injuries, or complications—that directly caused the death. DO NOT enter terminal events such as cardiac arrest, respiratory arrest, or ventricular fibrillation without showing the etiology. DO NOT ABBREVIATE. Enter only one cause on a line. Add additional lines if necessary. IMMEDIATE CAUSE (Final disease or condition) a. _____ resulting in death) Due to (or as a consequence of): Sequentially list conditions, b. _____ if any, leading to the cause Due to (or as a consequence of): listed on line a. Enter the <b>UNDERLYING CAUSE</b> c. _____ (disease or injury that Due to (or as a consequence of): initiated the events resulting in death) <b>LAST</b> d. _____ <input type="checkbox"/> <b>CAUSE OF DEATH (See instructions and examples)</b> Rupture of myocardium Acute myocardial infarction Coronary artery thrombosis Atherosclerotic coronary artery disease		Approximate interval: Onset to death _____ _____ _____ _____ Minutes 6 days 5 years 7 years
<b>PART II.</b> Enter other significant conditions contributing to death but not resulting in the underlying cause given in PART I. Diabetes, Chronic obstructive pulmonary disease, smoking		<b>33. WAS AN AUTOPSY PERFORMED?</b> Yes No <b>34. WERE AUTOPSY FINDINGS AVAILABLE TO COMPLETE THE CAUSE OF DEATH?</b> Yes No
<b>35. DID TOBACCO USE CONTRIBUTE TO DEATH?</b> Yes Probably No Unknown	<b>36. IF FEMALE:</b> Not pregnant within past year Pregnant at time of death Not pregnant, but pregnant within 42 days of death Not pregnant, but pregnant 43 days to 1 year before death Unknown if pregnant within the past year	<b>37. MANNER OF DEATH</b> Natural Homicide Accident Pending Investigation Suicide Could not be determined

<p>32. <b>PART I.</b> Enter the chain of events—diseases, injuries, or complications—that directly caused the death. DO NOT enter terminal events such as cardiac arrest, respiratory arrest, or ventricular fibrillation without showing the etiology. DO NOT ABBREVIATE. Enter only one cause on a line. Add additional lines if necessary. IMMEDIATE CAUSE (Final disease or condition)</p> <p>a. _____ _____ resulting in death) Due to (or as a consequence of): Sequentially list conditions,</p> <p>b. _____ _____ if any, leading to the cause Due to (or as a consequence of): listed on line a. Enter the <b>UNDERLYING CAUSE</b></p> <p>c. _____ _____ (disease or injury that Due to (or as a consequence of): initiated the events resulting in death) <b>LAST</b></p> <p>d. _____ _____ <input type="checkbox"/> <b>CAUSE OF DEATH (See instructions and examples)</b> Acute renal failure Hyperosmolar nonketotic coma Diabetes mellitus, noninsulin dependent</p>		<p>Approximate interval: Onset to death</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____ 5 days 8 weeks 15 years</p>
<p><b>PART II.</b> Enter other significant conditions contributing to death but not resulting in the underlying cause given in PART I.</p>		<p>33. WAS AN AUTOPSY PERFORMED? Yes No</p> <p>34. WERE AUTOPSY FINDINGS AVAILABLE TO COMPLETE THE CAUSE OF DEATH? Yes No</p>
<p>35. DID TOBACCO USE CONTRIBUTE TO DEATH? Yes Probably No Unknown</p>	<p>36. IF FEMALE: Not pregnant within past year Pregnant at time of death Not pregnant, but pregnant within 42 days of death Not pregnant, but pregnant 43 days to 1 year before death Unknown if pregnant within the past year</p>	<p>37. MANNER OF DEATH Natural Homicide Accident Pending Investigation Suicide Could not be determined</p>

### ITEM 32 - CAUSE OF DEATH

Take care to make the entry legible. Use a computer printer with high resolution, typewriter with good black ribbon and clean keys, or print legibly using permanent **black** ink in completing the cause-of-death section. **Do not abbreviate** conditions entered in section.

#### Part I (Chain of events leading directly to death)

- Only **one** cause should be entered on each line. Line a **MUST ALWAYS** have an entry. **DO NOT** leave blank. Additional lines may be added if necessary.
- If the condition on Line a resulted from an underlying condition, put the underlying condition on Line b, and so on, until the full sequence is reported. **ALWAYS** enter the **underlying cause of death** on the lowest used line in Part I.
- For each cause indicate the best estimate of the interval between the presumed onset and the date of death. The terms “unknown” or “approximately” may be used. General terms, such as minutes, hours, or days, are acceptable, if necessary. **DO NOT** leave blank. **U.S.**

Source: Department Of Health And Human Services, Centers for Disease Control and Prevention National Center for Health Statistics [http://www.cdc.gov/nchs/data/dvs/blue\\_form.pdf](http://www.cdc.gov/nchs/data/dvs/blue_form.pdf)

## APPENDIX C

### Mortality Data, Multiple Cause-of-Death Public-Use Data Files

Data File Description: Detail (1999-2002)

Data year

Residence of decedent:<sup>1</sup>

State<sup>2</sup>

County<sup>2,3</sup>

City<sup>3</sup>

Population size

Standard metropolitan statistical area<sup>2,3</sup>

Metropolitan and nonmetropolitan counties

Age at death:

Infants under 1 year (hours, days, months)

Infants ages 1 year and over (single years)

Day of week

Education (single years, 0-17)<sup>3</sup>

Hispanic origin

Hospital (including status of decedent) and other type  
of place of death

Industry or business<sup>3</sup>

Injury at work

Month of death

Marital status

Occupation (usual)<sup>4</sup>

Place of birth (State and eight categories outside of the United States)

Place of death (State,<sup>2,3</sup> county<sup>2,3</sup>)

Race (nine categories)<sup>5</sup>

Sex

State of birth

Manner of death

Activity code

Place of injury

Underlying cause of death:<sup>6</sup>

Each cause

Multiple condition codes: Entity axis, Record axis<sup>1</sup>

---

<sup>1</sup>Place of residence for decedents who were nonresidents of the United States has been coded to country of residence.

<sup>2</sup>Includes FIPS codes as well as NCHS codes.

<sup>3</sup>Includes data for areas with a population of 100,000 persons or more.

<sup>4</sup>Applicable only for those States transmitting information to NCHS.

<sup>5</sup>Beginning 1992, additional categories are available for some States.

<sup>6</sup>Causes of death for 1999-2002 were coded according to the International Classification of Diseases, Ninth Revision.

# Data File Description: Detail (1989-98)

Data year

Residence of decedent:<sup>1</sup>

State<sup>2</sup>

County<sup>2,3</sup>

City<sup>2</sup>

Population size

Standard metropolitan statistical area<sup>2,3</sup>

Metropolitan and nonmetropolitan counties

Age at death:

Infants under 1 year (hours, days, months)

Infants ages 1 year and over (single years)

Autopsy performed (1989-94)

Day of week

Education (single years, 0-17)<sup>4</sup>

Hispanic origin<sup>4</sup>

Hospital (including status of decedent) and other type of place of death

Industry or business<sup>5</sup>

Injury at work (beginning 1993)

Month of death

Marital status

Occupation (usual)<sup>5</sup>

Place of birth (State and eight categories outside of the United States)

Place of death (State,<sup>2,3</sup> county<sup>2,3</sup>)

Race (nine categories)<sup>6</sup>

Sex

State of birth

Underlying cause of death:<sup>7</sup>

Each cause

Multiple condition codes: Entity axis Record axis<sup>1</sup>

<sup>1</sup>Place of residence for decedents who were nonresidents of the United States has been coded to country of residence.

<sup>2</sup>Includes FIPS codes as well as NCHS codes.

<sup>3</sup>Includes data for areas with a population of 100,000 persons or more.

<sup>4</sup>Applicable only for those States having information on the certificate.

<sup>5</sup>Applicable only for those States transmitting information to NCHS.

<sup>6</sup>Beginning 1992, additional categories are available for some States.

<sup>7</sup>Causes of death for 1989-98 were coded according to the International Classification of Diseases, Ninth Revision.

Source: National Center for Health Statistics

## APPENDIX D

### Chapter Titles for the Ninth and Tenth Revisions, International Classification of Diseases

Chapter ICD–91 chapter titles (code range2)	ICD–101 chapter titles (code2)
I Infectious and parasitic diseases (001–139)	Infectious&parasitic diseases A00–B99)
II Neoplasms (140–239)	Neoplasms.(C00–D48)
III Endocrine, nutritional, metabolic diseases	Diseases of the blood and blood-forming organs and certain and immunity disorders (240–279) disorders involving the immune mechanism D50–D89)
IV Diseases blood/ blood-forming organs(280–289)	Endocrine, nutritional and metabolic diseases(E00–E90)
V Mental disorders (290–319)	Mental/behavioral disorders (F00–F99)
VI Diseases nervous/sense organs(320–389)	Diseases of nervous system (G00–G99)
VII Diseases of circulatory system 390–459)	Diseases of the eye/adnexa H00–H59)
VIII Diseases respiratory system (460–519)	Diseases ear/mastoid process H60–H95)
IX Diseases of digestive system (520–579)	Diseases of circulatory system.(I00–I99)
X Diseases genitourinary system (580–629)	Diseases respiratory system (J00–J99)
XI Complications pregnancy/birth (630–676)	Diseases digestive system K00–K93)
XII Diseases skin/subcutaneous (680–709)	Diseases skin/ subcutaneous (L00–L99)
XIII Diseases/musculoskeletal tissue (710–739)	Diseases musculoskeletal (M00–M99)
XIV Congenital anomalies (740–759)	Diseases of the genitourinary N00–N99)
XV Certain perinatal conditions (760–779)	Pregnancy/birth/puerperium (O00–O99)
XVI Symptoms, ill-defined (780–799)	Certain conditions perinatal (P00–P96)
XVII Injury and poisoning (800–999)	Congenital/ chromosomal (Q00–Q99)
XVIII - - - Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classifiedR00–R99)	
XIX - - - Injury, poisoning and certain other consequences of external causes S00–T98)	
XX - - - External causes of morbidity and mortality (V01–Y98)	
XXI - - - Factors influencing health status and contact with health services (Z00–Z99)	
- - - Supplementary classification of external causes of injury and poisoning (E800–E999) - - -	
- - - Supplementary classification of factors influencing health status and contact with health servicesV01–V82) - - -	
- - - Category not applicable.	

1ICD–9 is International Classification of Diseases, Ninth Revision, and ICD–10 is International Classification of Diseases, Tenth Revision.

2The fourth digits of the upper and lower limits of the code ranges are not shown.

Source: National Vital Statistics Report, Vol. 49, No. 2, May 18, 2001

## APPENDIX E

### Comparable category numbers for 113 selected causes of death according to the Ninth and Tenth Revisions, *International Classification of Diseases*

List

Number Cause of death Category codes	ICD-10	ICD-9
001 Salmonella infections. . . . .	A01-A02	002-003
002 Shigellosis and amebiasis. . . . .	A03,A06	004,006
003 Certain other intestinal infections. . . . .	A04,A07-A09	007-009
004 Tuberculosis. . . . .	A16-A19	010-018
005 Respiratory tuberculosis. . . . .	A16	010-012
006 Other tuberculosis . . . . .	A17-A19	013-018
007 Whooping cough . . . . .	A37	033
008 Scarlet fever and erysipelas . . . . .	A38,A46	034.1-035
009 Meningococcal infection . . . . .	A39	036
010 Septicemia. . . . .	A40-A41	038
011 Syphilis. . . . .	A50-A53	090-097
012 Acute poliomyelitis . . . . .	A80	045
013 Arthropod-borne viral encephalitis . . . . .	A83-A84,A85.2	062-064
014 Measles . . . . .	B05	055
015 Viral hepatitis . . . . .	B15-B19	070
016 Human immunodeficiency virus (HIV) disease . . . . .	B20-B24	042-044
017 Malaria . . . . .	B50-B54	084
018 Other/infectious& parasitic diseases & sequelae A00, A05, A20-A36,A42-A44,A48-A49,A54-, 054,056- A79,A81-A82,A85.0-A85.1,A85.8, 083, A86-B04,B06-B09,B25-B49,B55-B99	A00, 001,005,020-032, 037 039-041,046- 061,065-066,071- 085-088,098-134,136,139,771.3	
019 Malignant neoplasms. . . . .	C00-C97	140-208
020 Malignant neoplasms of lip, oral cavity & pharynx . . . . .	C00-C14	140-149
021 Malignant neoplasm of esophagus . . . . .	C15	150
022 Malignant neoplasm of stomach. . . . .	C16	151
023 Malignant neoplasms of colon, rectum and anus . . . . .	C18-C21	153-154
024 Malignant neoplasms of liver & intrahepatic bile ducts . . . . .	C22	155
025 Malignant neoplasm of pancreas . . . . .	C25	157
026 Malignant neoplasm of larynx . . . . .	C32	161
027 Malignant neoplasms of trachea, bronchus and lung. . . . .	C33-C34	162
028 Malignant melanoma of skin . . . . .	C43	172
029 Malignant neoplasm of breast . . . . .	C50	174-175
030 Malignant neoplasm of cervix uteri . . . . .	C53	180
031 Malignant neoplasms of corpus uteri & uterus . . . . .	C54-C55	179,182
032 Malignant neoplasm of ovary . . . . .	C56	183.0
033 Malignant neoplasm of prostate . . . . .	C61	185

034 Malignant neoplasms of kidney and renal pelvis . . .C64–C65	189.0,189.1
035 Malignant neoplasm of bladder . . . . . C67	188
036 Malignant neoplasms of meninges, brain CNS . . . C70–C72	191–
192	
037 Malignant neoplasms of lymphoid, hematopoietic. . C81–C96	200–208
038 Hodgkin's disease. . . . . C81	201
039 Non-Hodgkin's lymphoma . . . . . C82–C85	200,202
040 Leukemia. . . . . C91–C95	204–208
041 Multiple myeloma & immunoproliferative neoplasmC88,C90	203
042 Other malignant neoplasms/lymphoid/hematopoieticC96 - - -	
043 All other and unspecified malignant neoplasms . . . C17,C23–C24,	152,156
C26–C31,C37–C41,C44–C49,	158–160,163–171,173,181,183.2–
C51–C52,C57–C60,C62–C63,C66,C68–	184,186–187,189.2–190,193–199
C69,C73–C80,C97	
044 In situ neoplasms, benign neoplasms . . . . . D00–D48	210–239
045 Anemias . . . . .D50–D64	280–285
046 Diabetes mellitus . . . . . E10–E14	250
047 Nutritional deficiencies . . . . . E40–E64	260–269
048 Malnutrition . . . . . E40–E46	260–263
049 Other nutritional deficiencies . . . . . E50–E64	264–269
050 Meningitis . . . . . G00,G03	320–322
051 Parkinson's disease . . . . . G20–G21	332
052 Alzheimer's disease . . . . . G30	331.0
053 Major cardiovascular diseases . . . . . I00–I78	390–434,436–448
054 Diseases of heart . . . . . I00–I09,I11,I13,I20–I51	390–98,402,404,410–429
055 Acute/chronic rheumatic heart diseases . . . . . I00–I09	390–398
056 Hypertensive heart disease . . . . . I11	402
057 Hypertensive heart and renal disease . . . . . I13	404
058 Ischemic heart diseases . . . . . I20–I25	410–414,429.2
059 Acute myocardial infarction . . . . . I21–I22	410
060 Other acute ischemic heart diseases . . . . . I24	411
061 Other forms of chronic ischemic heart disease . . I20,I25	412–414,429.2
062 Atherosclerotic cardiovascular disease, so described. I25.0	429.2
063 All other forms of chronic ischemic heart disease. I20,I25.1–I25.9	412–414
064 Other heart diseases . . . . . I26–I51	415–429.1,429.3–429.9
065 Acute and subacute endocarditis . . . . . I33	421
066 Diseases of pericardium and acute myocarditis . . I30–I31,I40	
420,422–423	
067 Heart failure. . . . .I50	428
068 All other forms of heart disease . . I26–I28,I34–I38,I42–I49,I51	415–417,424–427,
	429.0–429.1,429.3–429.9
069 Essential hypertension/hypertensive renal disease I10,I12	401,403
070 Cerebrovascular diseases . . . . .I60–I69	430–434,436–438
071 Atherosclerosis . . . . .I70	440
072 Other diseases of circulatory system. . . . . I71–I78	441–448
073 Aortic aneurysm and dissection . . . . .I71	441

074 Other diseases of arteries, arterioles and capillaries I72–I78	442–448
075 Other disorders of circulatory system . . . . . I80–I99	451–459
076 Influenza and pneumonia . . . . . J10–J18	480–487
077 Influenza . . . . . J10–J11	487
078 Pneumonia . . . . . J12–J18	480–486
079 Other acute lower respirator. . . . . y infections. . . . . J20–J22	466
080 Acute bronchitis and bronchiolitis . . . . . J20–J21	466
081 Unspecified acute lower respiratory infection . . . . J22 - - -	
082 Chronic lower respiratory diseases. . . . . J40–J47	490–494,496
083 Bronchitis, chronic and unspecified . . . . . J40–J42	490–491
084 Emphysema . . . . . J43	492
085 Asthma . . . . . J45–J46	493
086 Other chronic lower respiratory diseases. . . . . J44,J47	494,496
087 Pneumoconioses and chemical effects . . . . . J60–J66,J68	500–506
088 Pneumonitis due to solids and liquids. . . . . J69	507
089 Other diseases of respiratory system . J00–J06,J30–J39,J67,J70–J98	034.0,460–465,470–478,495,508–519
090 Peptic ulcer . . . . . K25–K28	531–534
091 Diseases of appendix . . . . . K35–K38	540–543
092 Hernia . . . . . K40–K46	550–553
093 Chronic liver disease and cirrhosis . . . . . K70,K73–K74	571
094 Alcoholic liver disease . . . . . K70	571.0–571.3
095 Other chronic liver disease and cirrhosis. . . . . K73–K74	571.4–571.9
096 Cholelithiasis and other disorders of gallbladder. . K80–K82	574–575
097 Nephritis, nephrotic syndrome and nephrosis . . . N00–N07,	580–589
N17–N19,N25–N27	
098 Acute&rapidly progressive nephritic syndrome. . N00–N01,N04	580–581
099 Chronic glomerulonephritis, nephritisNOS N02–N03,N05–N07,N26	582–583,587
100 Renal failure . . . . . N17–N19	584–586
101 Other disorders of kidney. . . . . N25,N27	588–589
102 Infections of kidney. . . . . N10–N12,N13.6,N15.1	590
103 Hyperplasia of prostate . . . . . N40	600
104 Inflammatory diseases of female pelvic organs . . . N70–N76	614–
616	
105 Pregnancy, childbirth and the puerperium. . . . . O00–O99	630–676
106 Pregnancy with abortive outcome . . . . . O00–O07	630–639
107 Other complications/pregnancy, childbirth/puerperium O10–O99	640–676
108 Certain conditions originating/perinatal period . . P00–P96	760–771.2,771.4–779
109 Congenital malformations/chromosomal abnormalities Q00–Q99	740–759
110 Symptoms/ abnormal clinical/laboratory NOS . . . . R00–R99	780–799
111 All other diseases (Residual) . . . . . Residual	Residual
112 Accidents (unintentional injuries) . . V01–X59,Y85–Y86	E800–E869,E880–E929
113 Transport accidents . . . . . V01–V99,Y85	E800–E848,E929.0,E929.1
114 Motor vehicle accide V02–V04,V09.0,V09.2,,V12–V14,V19.0–	E810–E825



V19.2,V19.4–V19.6,V20–V79,V80.3–V80.5,V81.0–V81.1,V82.0–V82.1,V83–V86,V87.0–V87.8,V88.0–V88.8,V89.0,V89.2	
115 Other land transport accidents 01,V05–V06,V09.1, V09.3–V09.9,V10–V11,V15–V18,V19.3,V19.8–V19.9, V80.0–V80.2,V80.6–V80.9,V81.2–V81.9,V82.2– V82.9,V87.9,V88.9,V89.1,V89.3,V89.9	E800–E807,E826–E829
116 Water, air/space,/NOS transport accidents . . V90–V99,Y85	E830– E848,E929.0,E929.1
117 Nontransport accidents . . . . . W00–X59,Y86	E850–E869,E880– E928,E929.2–E929.9
118 Falls. . . . . W00–W19	E880–E888
119 Accidental discharge of firearms . . . . . W32–W34	E922
120 Accidental drowning and submersion . . . . . W65–W74	E910
121 Accidental exposure to smoke, fire and flames . . . X00–X09	E890–E899
122 Accidental poisoning/noxious substances. . . . . X40–X49	E850–E869,E924.1
123 NOS nontransport accidents and their sequelae. W20–W31, W35–W64,W75–W99,X10–, X39,X50–X59,Y86	E900–E909 E911–E921,E923– E924.0,E924.8– E928,E929.2–E929.9
124 Intentional self-harm (suicide). . . . . X60–X84,Y87.0	E950–E959
125 Intentional self-harm (suicide) by discharge of firearms .X72–X74	E955.0–E955.4
126 Intentional self-harm (suicide)NOS. . . . . X60–X71,X75–X84,Y87.0	E950–E954, E955.5–E959
127 Assault (homicide) . . . . . X85–Y09,Y87.1	E960–E969
128 Assault (homicide) by discharge of firearms. . . . . X93–X95	E965.0–E965.4
129 Assault (homicide) NOS sequelae . . . . X85–X92,X96–Y09,Y87.1	E960–E964, E965.5–E969
130 Legal intervention. . . . . Y35,Y89.0	E970–E978
131 Events of undetermined intent . . . . . Y10–Y34,Y87.2,Y89.9	E980–E989
132 Discharge of firearms, undetermined intent . . . . . Y22–Y24	E985.0–E985.4
133 Other/unspecified events and their sequelae. . . . Y10–Y21,Y25–Y34,Y87.2,Y89.9	E980–E984,E985.5–E989
134 Operations of war and their sequelae . . . . Y36,Y89.1	E990–E999
135 Complications of medical and surgical care . . . . Y40–Y84,Y88	E870–E879, E930–E949

- - - No comparable category classified by ICD–9 exists.

1ICD–10 is *International Classification Diseases*, Tenth Revision, and ICD–9 is *International Classification of Diseases*, Ninth Revision.

Source: National Vital Statistics Report, Vol. 49, No. 2, May 18, 2001

## APPENDIX F

**Deaths, Percent Of Total Deaths, And Death Rates For The 15 Leading Causes Of  
Death In 5-Year Age Groups, Aged 60-65 All Races And Both Sexes: United States,  
2002**

Rates are not shown for age groups over 85 years because population figures are not available for these age groups. [Rates per 100,000 population in specified group. Percent of Cause of death (Based on the Tenth Revision, International Classification of Diseases)]

## All races, both sexes, all ages

...	All causes	2,443,387	100.0	847.3
1	Diseases of heart (I00-I09,I11,I13,I20-I51)	696,947	28.5	241.7
2	Malignant neoplasms (C00-C97)	557,271	22.8	193.2
3	Cerebrovascular diseases (I60-I69)	162,672	6.7	56.4
4	Chronic lower respiratory diseases (J40-J47)	124,816	5.1	43.3
5	Accidents (unintentional injuries) (V01-X59,Y85-Y86)	106,742	4.4	37.0
6	Diabetes mellitus (E10-E14)	73,249	3.0	25.4
7	Influenza and pneumonia (J10-J18)	65,681	2.7	22.8
8	Alzheimer's disease (G30)	58,866	2.4	20.4
9	Nephritis, nephrotic syndrome (N00-N07,N17-N19,N25-N27)	40,974	1.7	14.2
10	Septicemia (A40-A41)	33,865	1.4	11.7
11	Intentional self-harm (suicide) (*U03,X60-X84,Y87.0)	31,655	1.3	11.0
12	Chronic liver disease and cirrhosis (K70,K73-K74)	27,257	1.1	9.5
13	Essential hypertension and hypertensive renal disease (I10,I12)	20,261	0.8	7.0
14	Assault (homicide) (*U01-*U02,X85-Y09,Y87.1)	17,638	0.7	6.1
15	Pneumonitis due to solids and liquids (J69)	17,593	0.7	6.1
...	All other causes (Residual)	407,900	16.7	141.5

## All races, both sexes, 60-64 years

...	All causes	137,901	100.0	1,187.7
1	Malignant neoplasms (C00-C97)	51,904	37.6	447.0
2	Diseases of heart (I00-I09,I11,I13,I20-I51)	35,339	25.6	304.4
3	Chronic lower respiratory diseases (J40-J47)	7,060	5.1	60.8
4	Cerebrovascular diseases (I60-I69)	5,604	4.1	48.3
5	Diabetes mellitus (E10-E14)	5,512	4.0	47.5

6	Accidents (unintentional injuries) (V01-X59,Y85-Y86)	3,788	2.7	32.6
7	Chronic liver disease and cirrhosis (K70,K73-K74)	2,839	2.1	24.5
8	Nephritis, nephrotic syndrome and nephrosis (N00-N07,N17-N19,N25-N27)	1,950	1.4	16.8
9	Septicemia (A40-A41)	1,861	1.3	16.0
10	Influenza and pneumonia (J10-J18)	1,716	1.2	14.8
11	Intentional self-harm (suicide) (*U03,X60-X84,Y87.0)	1,432	1.0	12.3
12	Aortic aneurysm and dissection (I71)	862	0.6	7.4
13	Essential (primary) hypertension and hypertensive renal disease (I10,I12)	845	0.6	7.3
14	In situ neoplasms, benign neoplasms and neoplasms of uncertain or unknown beha 632		0.5	5.4
15	Human immunodeficiency virus (HIV) disease (B20-B24)	462	0.3	4.0
...	All other causes (Residual)	16,095	11.7	138.6

---

All races, both sexes, 65-69 years

...	All causes	175,591	100.0	1,832.7
1	Malignant neoplasms (C00-C97)	63,598	36.2	663.8
2	Diseases of heart (I00-I09,I11,I13,I20-I51)	45,948	26.2	479.6
3	Chronic lower respiratory diseases (J40-J47)	11,405	6.5	119.0
4	Cerebrovascular diseases (I60-I69)	8,026	4.6	83.8
5	Diabetes mellitus (E10-E14)	7,249	4.1	75.7
6	Accidents (unintentional injuries) (V01-X59,Y85-Y86)	3,672	2.1	38.3
7	Nephritis, nephrotic syndrome and nephrosis (N00-N07,N17-N19,N25-N27)	2,820	1.6	29.4
8	Chronic liver disease and cirrhosis (K70,K73-K74)	2,670	1.5	27.9
9	Septicemia (A40-A41)	2,589	1.5	27.0
10	Influenza and pneumonia (J10-J18)	2,420	1.4	25.3
11	Aortic aneurysm and dissection (I71)	1,404	0.8	14.7
12	Intentional self-harm (suicide) (*U03,X60-X84,Y87.0)	1,197	0.7	12.5
13	Essential (primary) hypertension and	1,126	0.6	11.8

3	hypertensive renal disease (I10,I12)			
1	Alzheimer's disease (G30)	939	0.5	9.8
4				
1	In situ neoplasms, benign neoplasms and	928	0.5	9.7
5	neoplasms of uncertain or unknown beha			
...	All other causes (Residual)	19,600	11.2	204.6

All races, both sexes, 70-74 years

			100.	2,845.
All causes	247,399	0		9
1 Malignant neoplasms (C00-C97)	81,159	32.8		933.6
2 Diseases of heart (I00-I09,I11,I13,I20-I51)	66,599	26.9		766.1
3 Chronic lower respiratory diseases (J40-J47)	18,383	7.4		211.5
4 Cerebrovascular diseases (I60-I69)	13,966	5.6		160.7
5 Diabetes mellitus (E10-E14)	9,460	3.8		108.8
6 Influenza and pneumonia (J10-J18)	4,427	1.8		50.9
7 Accidents (unintentional injuries) (V01-X59,Y85-Y86)	4,414	1.8		50.8
8 Nephritis, nephrotic syndrome and nephrosis (N00-N07,N17-N19,N25-N27)	4,344	1.8		50.0
9 Septicemia (A40-A41)	3,747	1.5		43.1
1 Chronic liver disease and cirrhosis (K70,K73-K74)	2,711	1.1		31.2
0 Alzheimer's disease (G30)	2,663	1.1		30.6
1				
1 Aortic aneurysm and dissection (I71)	2,078	0.8		23.9
2				
1 Essential (primary) hypertension and	1,796	0.7		20.7
3 hypertensive renal disease (I10,I12)				
1 Parkinson's disease (G20-G21)	1,600	0.6		18.4
4				
1 In situ neoplasms, benign neoplasms and	1,420	0.6		16.3
5 neoplasms of uncertain or unknown beha				
...	All other causes (Residual)	28,632	11.6	329.4

All races, both sexes, 75-79 years

			100.	4,449.
...	All causes	330,140	0	1
1	Diseases of heart (I00-I09,I11,I13,I20-I51)	94,844	28.7	1,278.2
2	Malignant neoplasms (C00-C97)	89,565	27.1	1,207.0

3	Chronic lower respiratory diseases (J40-J47)	24,495	7.4	330.1
4	Cerebrovascular diseases (I60-I69)	22,911	6.9	308.8
5	Diabetes mellitus (E10-E14)	11,703	3.5	157.7
6	Influenza and pneumonia (J10-J18)	7,905	2.4	106.5
7	Alzheimer's disease (G30)	7,078	2.1	95.4
8	Nephritis, nephrotic syndrome and nephrosis (N00-N07,N17-N19,N25-N27)	6,338	1.9	85.4
9	Accidents (unintentional injuries) (V01-X59,Y85-Y86)	5,938	1.8	80.0
1	Septicemia (A40-A41)	5,195	1.6	70.0
0				
1	Parkinson's disease (G20-G21)	3,368	1.0	45.4
1				
1	Aortic aneurysm and dissection (I71)	2,779	0.8	37.5
2				
1	Essential (primary) hypertension and hypertensive renal disease (I10,I12)	2,592	0.8	34.9
3				
1	Chronic liver disease and cirrhosis (K70,K73-K74)	2,412	0.7	32.5
4				
1	Pneumonitis due to solids and liquids (J69)	2,323	0.7	31.3
5				
...	All other causes (Residual)	40,694	12.3	548.4

All races, both sexes, 80-84 years

			100.	7,103.
...	All causes	377,514	0	8
1	Diseases of heart (I00-I09,I11,I13,I20-I51)	118,737	31.5	2,234.3
2	Malignant neoplasms (C00-C97)	77,497	20.5	1,458.3
3	Cerebrovascular diseases (I60-I69)	31,978	8.5	601.7
4	Chronic lower respiratory diseases (J40-J47)	24,746	6.6	465.7
5	Alzheimer's disease (G30)	13,057	3.5	245.7
6	Influenza and pneumonia (J10-J18)	12,079	3.2	227.3
7	Diabetes mellitus (E10-E14)	11,579	3.1	217.9
8	Nephritis, nephrotic syndrome and nephrosis (N00-N07,N17-N19,N25-N27)	7,558	2.0	142.2
9	Accidents (unintentional injuries) (V01-X59,Y85-Y86)	6,966	1.8	131.1
1	Septicemia (A40-A41)	5,815	1.5	109.4
0				
1	Parkinson's disease (G20-G21)	4,771	1.3	89.8
1				
1	Pneumonitis due to solids and liquids (J69)	3,577	0.9	67.3
2				

1	Essential (primary) hypertension and	3,546	0.9	66.7
3	hypertensive renal disease (I10,I12)			
1	Aortic aneurysm and dissection (I71)	2,739	0.7	51.5
4				
1	Atherosclerosis (I70)	2,541	0.7	47.8
5				
...	All other causes (Residual)			

## All races, both sexes, 85-89 years

		345,38		
...	All causes	5	100.0	...
1	Diseases of heart (I00-I09,I11,I13,I20-I51)	119,56	34.6	...
		5		...
2	Malignant neoplasms (C00-C97)	50,298	14.6	...
3	Cerebrovascular diseases (I60-I69)	33,197	9.6	...
4	Chronic lower respiratory diseases (J40-J47)	17,971	5.2	...
5	Alzheimer's disease (G30)	16,267	4.7	...
6	Influenza and pneumonia (J10-J18)	13,977	4.0	...
7	Diabetes mellitus (E10-E14)	8,797	2.5	...
8	Nephritis, nephrotic syndrome and nephrosis (N00-N07,N17-N19,N25-N27)	6,959	2.0	...
9	Accidents (unintentional injuries) (V01-X59,Y85-Y86)	6,507	1.9	...
10	Septicemia (A40-A41)	5,026	1.5	...
11	Pneumonitis due to solids and liquids (J69)	4,100	1.2	...
12	Parkinson's disease (G20-G21)	3,959	1.1	...
13	Essential (primary) hypertension and hypertensive renal disease (I10,I12)	3,801	1.1	...
14	Atherosclerosis (I70)	3,090	0.9	...
15	In situ neoplasms, benign neoplasms and neoplasms of uncertain or unknown beha	2,052	0.6	...
...	All other causes (Residual)	49,819	14.4	...

## All races, both sexes, 90-94 years

		228,58		
...	All causes	7	100.0	...
1	Diseases of heart (I00-I09,I11,I13,I20-I51)	86,848	38.0	...
2	Cerebrovascular diseases (I60-I69)	23,110	10.1	...
3	Malignant neoplasms (C00-C97)	22,323	9.8	...
4	Alzheimer's disease (G30)	12,267	5.4	...
5	Influenza and pneumonia (J10-J18)	11,490	5.0	...

6	Chronic lower respiratory diseases (J40-J47)	8,508	3.7	...
7	Nephritis, nephrotic syndrome and nephrosis (N00-N07,N17-N19,N25-N27)	4,450	1.9	...
8	Diabetes mellitus (E10-E14)	4,375	1.9	...
9	Accidents (unintentional injuries) (V01-X59,Y85-Y86)	4,255	1.9	...
10	Pneumonitis due to solids and liquids (J69)	2,995	1.3	...
11	Septicemia (A40-A41)	2,988	1.3	...
12	Essential (primary) hypertension and hypertensive renal disease (I10,I12)	2,915	1.3	...
13	Atherosclerosis (I70)	2,802	1.2	...
14	Parkinson's disease (G20-G21)	1,781	0.8	...
15	In situ neoplasms, benign neoplasms and neoplasms of uncertain or unknown beha	1,162	0.5	...
...	All other causes (Residual)	36,318	15.9	...

All races, both sexes, 95-99 years

...	All causes	88,282	100.0	...
1	Diseases of heart (I00-I09,I11,I13,I20-I51)	35,664	40.4	...
2	Cerebrovascular diseases (I60-I69)	8,521	9.7	...
3	Malignant neoplasms (C00-C97)	5,772	6.5	...
4	Influenza and pneumonia (J10-J18)	5,172	5.9	...
5	Alzheimer's disease (G30)	5,140	5.8	...
6	Chronic lower respiratory diseases (J40-J47)	2,430	2.8	...
7	Accidents (unintentional injuries) (V01-X59,Y85-Y86)	1,595	1.8	...
8	Nephritis, nephrotic syndrome and nephrosis (N00-N07,N17-N19,N25-N27)	1,549	1.8	...
9	Atherosclerosis (I70)	1,416	1.6	...
10	Diabetes mellitus (E10-E14)	1,357	1.5	...
11	Essential (primary) hypertension and hypertensive renal disease (I10,I12)	1,276	1.4	...
12	Pneumonitis due to solids and liquids (J69)	1,219	1.4	...
13	Septicemia (A40-A41)	1,111	1.3	...
14	Parkinson's disease (G20-G21)	440	0.5	...
15	In situ neoplasms, benign neoplasms and neoplasms of uncertain or unknown beha	366	0.4	...
...	All other causes (Residual)	15,254	17.3	...

## All races, both sexes, 100 years and over

...	All causes	18,822	100.0	...
1	Diseases of heart (I00-I09,I11,I13,I20-I51)	8,096	43.0	...
2	Cerebrovascular diseases (I60-I69)	1,584	8.4	...
3	Influenza and pneumonia (J10-J18)	1,356	7.2	...
4	Alzheimer's disease (G30)	878	4.7	...
5	Malignant neoplasms (C00-C97)	789	4.2	...
6	Atherosclerosis (I70)	415	2.2	...
7	Chronic lower respiratory diseases (J40-J47)	375	2.0	...
8	Nephritis, nephrotic syndrome and nephrosis (N00-N07,N17-N19,N25-N27)	298	1.6	...
9	Accidents (unintentional injuries) (V01-X59,Y85-Y86)	294	1.6	...
10	Essential (primary) hypertension and hypertensive renal disease (I10,I12)	293	1.6	...
11	Pneumonitis due to solids and liquids (J69)	229	1.2	...
12	Septicemia (A40-A41)	199	1.1	...
13	Diabetes mellitus (E10-E14)	195	1.0	...
14	Nutritional deficiencies (E40-E64)	93	0.5	...
15	Anemias (D50-D64)	78	0.4	...
...	All other causes (Residual)	3,650	19.4	...

Source: CDC/NCHS, National Vital Statistics System

Rank based on number of deaths

\* Figure does not meet standards of reliability or precision... Category not applicable ---

Data not available



## APPENDIX G

Table G.1.A Frequency of Decedents Over 60 by Top Causes of Mortality United States 1998-2002

	Heart	Cancer	Cardio Vascular	Pneumonia	AD	Diabetes	Nephritis	Hyper Tension	Septicemia	Suicide
60-64	143,053	250,518	37,953	8,839	3,227	26,380	9,273	5,928	7,968	6,482
65-69	195,129	325,401	58,243	13,547	7,865	35,815	14,503	7,816	11,487	5,937
70-74	286,749	416,225	98,534	25,195	20,360	47,807	22,371	11,374	16,817	6,331
75-79	400,165	442,506	154,664	42,975	46,243	56,811	31,684	15,935	23,108	6,235
80-84	477,680	367,056	198,640	63,876	73,798	53,816	36,752	19,860	25,367	4,978
85-89	482,791	239,842	203,509	74,211	80,986	40,225	34,900	20,703	23,311	2,894
90-94	345,003	104,803	140,128	60,011	55,817	20,224	22,579	15,118	14,277	1,001
95-99	142,570	27,286	53,632	27,056	21,098	6,209	8,309	6,399	5,262	176
100-104	29,559	3,448	9,839	6,406	3,427	871	1,498	1,368	882	11
105-109	3,009	229	875	738	274	67	155	120	81	0
110-114	144	14	53	48	8	3	13	12	5	0
115-119	12	1	2	4	0	0	0	1	0	0
120+	2	2	0	2	0	0	0	0	0	0
Total	2,505,866	2,177,331	956,072	322,908	313,103	288,228	182,037	104,634	128,565	34,045
9,643,607	26.0%	22.6%	9.9%	3.3%	3.2%	3.0%	1.9%	1.1%	1.3%	0.4%
(Total includes more than top causes)										

Table G.1.B: Frequency Distribution of Decedents, Over Age 60, by Age and Ethnicity, United States 1998-2002

Age	60-64	65-69	70-74	75-79	80-84	85-89	90-94	95-99	100-4	105+	Total
Mexican	19,367	24,458	30,632	32,225	26,626	22,028	13,859	5,790	111	15	175,111
	2.87%	2.68%	2.36%	1.93%	1.46%	1.32%	1.28%	1.38%	1.36%	3.05%	1.83%
PuertoRican	5,711	6,490	6,982	7,034	6,311	5,173	2,960	1,112	40	10	41,823
	0.85%	0.71%	0.54%	0.42%	0.35%	0.31%	0.27%	0.26%	0.49%	2.03%	0.44%
Cuban	3,211	4,576	6,441	7,849	8,687	8,767	6,072	2,335	31	3	47,972
	0.48%	0.50%	0.50%	0.47%	0.48%	0.53%	0.56%	0.56%	0.38%	0.61%	0.50%
Central&South	3,441	4,105	4,629	4,501	4,247	3,456	2,163	802	15	2	27,361
American	0.51%	0.45%	0.36%	0.27%	0.23%	0.21%	0.20%	0.19%	0.18%	0.41%	0.29%
Other	3,662	4,795	6,209	7,058	6,961	6,182	4,375	1,824	29	3	41,098
Hispanic	0.54%	0.52%	0.48%	0.42%	0.38%	0.37%	0.40%	0.43%	0.36%	0.61%	0.43%
NonHispanic	515,457	721,412	1,065,132	1,418,252	1,593,420	1,477,606	968,511	371,078	6,323	208	8,137,399
White	76.32%	78.93%	82.14%	84.87%	87.45%	88.64%	89.20%	88.30%	77.51%	42.28%	85.11%
NonHispanic	105,175	124,668	147,365	160,356	143,864	117,126	72,035	31,131	1,426	218	903,364
Black	15.57%	13.64%	11.36%	9.60%	7.90%	7.03%	6.63%	7.41%	17.48%	44.31%	9.45%
NonHispanic	16,627	20,402	25,270	28,974	27,177	22,318	13,048	5,022	151	31	159,020
Other	2.46%	2.23%	1.95%	1.73%	1.49%	1.34%	1.20%	1.19%	1.85%	6.30%	1.66%
Hispanic	2,720	3,033	4,070	4,774	4,775	4,372	2,771	1,160	32	2	27,709
Unknown	0.40%	0.33%	0.31%	0.29%	0.26%	0.26%	0.26%	0.28%	0.39%	0.41%	0.29%
Total	675,371	913,939	1,296,730	1,671,023	1,822,068	1,667,028	1,085,794	420,254	8,158	492	9,560,857

Table G.1.C Frequency of Decedents Aged 60 and Over by Ethnicity, Mortality by All Causes, AD and AD as Any Condition in the United States 1998-2002

	Total Causes					Total AD					AD as Any				
	Non		Non		Total	Non		Non		Total	Non		Non		Total
	Other	Hispanic	White	Black		Other	Hispanic	White	Black		Other	Hispanic	White	Black	
60-64	16,627	38,112	515,457	105,175	675,371	54	140	2,819	214	3,227	85	214	3,913	407	4,619
65-69	20,402	47,457	721,412	124,668	913,939	123	358	6,875	509	7,865	193	544	10,044	955	11,736
70-74	25,270	58,963	1,065,132	147,365	1,296,730	239	733	18,151	1,237	20,360	428	1,184	27,784	2,277	31,673
75-79	28,974	63,441	1,418,252	160,356	1,671,023	419	1,401	41,859	2,564	46,243	786	2,309	65,293	4,579	72,967
80-84	27,177	57,607	1,593,420	143,864	1,822,068	639	1,773	67,938	3,448	73,798	1,168	2,960	106,086	6,230	116,444
85-89	22,318	49,978	1,477,606	117,126	1,667,028	673	1,909	74,697	3,707	80,986	1,198	3,201	116,319	6,395	127,113
90-94	13,048	32,200	968,511	72,035	1,085,794	418	1,277	51,710	2,412	55,817	723	2,090	78,610	4,114	85,537
95-99	5,022	13,023	371,078	31,131	420,254	151	500	19,299	1,148	21,098	279	816	28,378	1,828	31,301
100-104	1,124	2,416	71,031	8,179	82,750	42	79	3,024	282	3,427	61	119	4,326	417	4,923
105-109	151	258	6,323	1,426	8,158	5	8	222	39	274	8	12	309	62	391
110-114	27	31	199	187	444	0	0	3	4	7	0	1	4	6	11
115-119	3	3	9	26	41	0	0	0	1	1	0	0	1	2	3
120+	1	1	0	5	7	0	0	0	0	0	0	0	0	0	0
Total	160,144	363,490	8,208,430	911,543	9,643,607	2,763	8,178	286,597	15,565	313,103	4,929	13,450	441,067	27,272	486,718
%	1.7%	3.8%	85.1%	9.5%		0.9%	2.6%	91.5%	5.0%		1.0%	2.8%	90.6%	5.6%	

Table G.1.D Frequency of Decedents by Age and Sex, All Causes, AD and AD as Any,  
in Decedents Over Age 60, United States 1998-2002

Age	All Causes			AD			AD as Any		
	Male	Female	Total	Female	Male	Total	Female	Male	Total
60-64	276,263	399,108	675,371	1,535	1,692	3,227	2,128	2,491	4,619
65-69	388,393	525,546	913,939	3,635	4,230	7,865	5,207	6,529	11,736
70-74	579,548	717,182	1,296,730	9,840	10,520	20,360	14,834	16,839	31,673
75-79	809,460	861,563	1,671,023	24,208	22,035	46,243	37,156	35,811	72,967
80-84	982,377	839,691	1,822,068	43,067	30,731	73,798	66,292	50,152	116,444
85-89	1,026,576	640,452	1,667,028	53,768	27,218	80,986	82,363	44,750	127,113
90-94	764,922	320,872	1,085,794	42,304	13,513	55,817	63,612	21,925	85,537
95-99	328,264	91,990	420,254	17,745	3,353	21,098	26,041	5,260	31,301
100-104	69,475	13,275	82,750	3,082	345	3,427	4,404	519	4,923
105-109	7,157	1,001	8,158	256	18	274	363	28	391
110-114	385	59	444	7	0	7	11	0	11
115-119	31	10	41	1	0	1	3	0	3
120+	4	3	7	0	0	0	0	0	0
	5,232,855	4,410,752	9,643,607	199,448	113,655	313,103	302,414	184,304	486,718

Table G.1.E Frequency of Decedents Over Age 60 by Marital Status Mortality All Causes and AD,  
United States 1998-2002

Age	Total Causes				Total	AD				Total
	Single	Married	Divorced	Widowed		Single	Married	Divorced	Widowed	
60-64	46,152	311,662	111,285	68,150	675,371	241	1,714	455	314	3,227
65-69	51,332	415,800	116,278	139,767	913,939	408	4,152	874	1,047	7,865
70-74	64,109	553,451	122,838	285,821	1,296,730	841	10,016	1,539	4,078	20,360
75-79	73,189	636,022	113,381	505,620	1,671,023	1,657	20,109	2,505	13,077	46,243
80-84	74,677	563,462	86,715	733,442	1,822,068	2,515	25,417	2,962	28,718	73,798
85-89	70,676	356,877	58,442	847,532	1,667,028	2,949	18,833	2,661	41,280	80,986
90-94	50,227	133,118	29,937	657,049	1,085,794	2,450	6,983	1,631	34,174	55,817
95-99	20,910	24,860	9,886	280,615	420,254	1,074	1,160	544	14,508	21,098
100-104	4,407	2,175	1,584	58,306	82,750	197	61	81	2,498	3,427
105-109	461	100	143	5,782	8,158	15	2	6	204	274
110-114	29	4	7	315	444	0	0	0	6	7
115-119	2	0	1	28	41	0	0	0	1	1
120+	1	0	1	4	7	0	0	0	0	0
	456,172	2,997,531	650,498	3,582,431	9,643,607	12,347	88,447	13,258	139,905	313,103
%	4.7%	31.1%	6.7%	37.1%		3.9%	28.2%	4.2%	44.7%	

(Total includes the not stated category)

Table G.1.F Frequency of Decedents Over Age 60 by Marital Status Mortality All Causes and AD as Any Condition, United States 1998-2002

	Total				AD as Any Condition					
	Single	Married	Divorced	Widowed	Total	Single	Married	Divorced	Widowed	Total
60-64	46,152	311,662	111,285	68,150	675,371	382	2,289	666	436	4,619
65-69	51,332	415,800	116,278	139,767	913,939	646	5,806	1,277	1,573	11,736
70-74	64,109	553,451	122,838	285,821	1,296,730	1,408	14,548	2,407	6,272	31,673
75-79	73,189	636,022	113,381	505,620	1,671,023	2,699	29,801	3,978	20,024	72,967
80-84	74,677	563,462	86,715	733,442	1,822,068	3,930	37,942	4,611	43,962	116,444
85-89	70,676	356,877	58,442	847,532	1,667,028	4,581	28,345	3,995	62,026	127,113
90-94	50,227	133,118	29,937	657,049	1,085,794	3,625	10,438	2,374	50,106	85,537
95-99	20,910	24,860	9,886	280,615	420,254	1,509	1,694	779	20,604	31,301
100-104	4,407	2,175	1,584	58,306	82,750	267	84	117	3,463	4,923
105-109	461	100	143	5,782	8,158	23	3	8	286	391
110-114	29	4	7	315	444	0	0	0	9	11
115-119	2	0	1	28	41	0	0	0	3	3
120+	1	0	1	4	7					
Total	456,172	2,997,531	650,498	3,582,431	9,643,607	19,070	130,950	20,212	208,764	486,718
%	4.7%	31.1%	6.7%	37.1%		3.9%	26.9%	4.2%	42.9%	

(Total includes the not stated category)

Table G.1.G Frequency of Decedents Over Aged 60, by Education ,  
Mortality by All Causes, United States 1998-2002

	Not stated	Elementary	High School	College	Total
60-64	49,752	79,513	370,379	175,727	675,371
65-69	65,285	132,950	490,843	224,861	913,939
70-74	89,496	214,772	689,390	303,072	1,296,730
75-79	112,260	297,261	876,684	384,818	1,671,023
80-84	121,725	380,218	922,656	397,469	1,822,068
85-89	115,266	416,518	783,015	352,229	1,667,028
90-94	79,337	313,363	451,724	241,370	1,085,794
95-99	33,229	138,239	158,299	90,487	420,254
100-104	7,462	30,726	28,423	16,139	82,750
105-109	883	3,370	2,521	1,384	8,158
110-114	64	228	103	49	444
115-119	11	25	4	1	41
120+	1	5	1	0	7
Total	674,771	2,007,188	4,774,042	2,187,606	9,643,607
%	7.0%	20.8%	49.5%	22.7%	

Table G.1.H: Frequency of Decedents Over Age 60:  
Education Level, Mortality by Cause of AD or AD as Any Condition

Education	AD	Other	ADANY	Other
Elementary	61,660 19.69%	1,945,528 20.85%	101,069 20.77%	1,906,119 20.82%
High School	146,858 46.90%	3,156,525 33.83%	185,835 38.18%	3,095,067 33.80%
College	84,617 27.03%	2,102,989 22.54%	126,279 25.95%	2,061,327 22.51%
Not stated	19,968 6.38%	654,803 7.02%	31,549 6.48%	643,222 7.02%
Total	313,103	9,330,504	486,718	9,156,889



Table G.1.I Frequency of Decedents Over Aged 60,  
By Metropolitan Status, Mortality by Cause of AD,  
United States 1998-2002

	AD	other
Metro	238,403	7,042,146
%	76.15%	75.55%
Nonmetro	74,655	2,279,098
%	23.85%	24.45%
Total	313,058	9,321,244

Table G.1.J Frequency of Decedents Over Aged 60,  
By Metropolitan Status, Mortality by Cause of AD :  
Any of the Conditions, United States 1998-2002

	ADANY	Other
Metro	367,280	6,913,269
%	75.47%	75.57%
Nonmetro	119,370	2,234,383
%	24.53%	24.43%
Total	486,650	9,147,652

Table G.1.K Frequency of Decedents Over Age 60 by Place of Death,  
United States 1998-2002

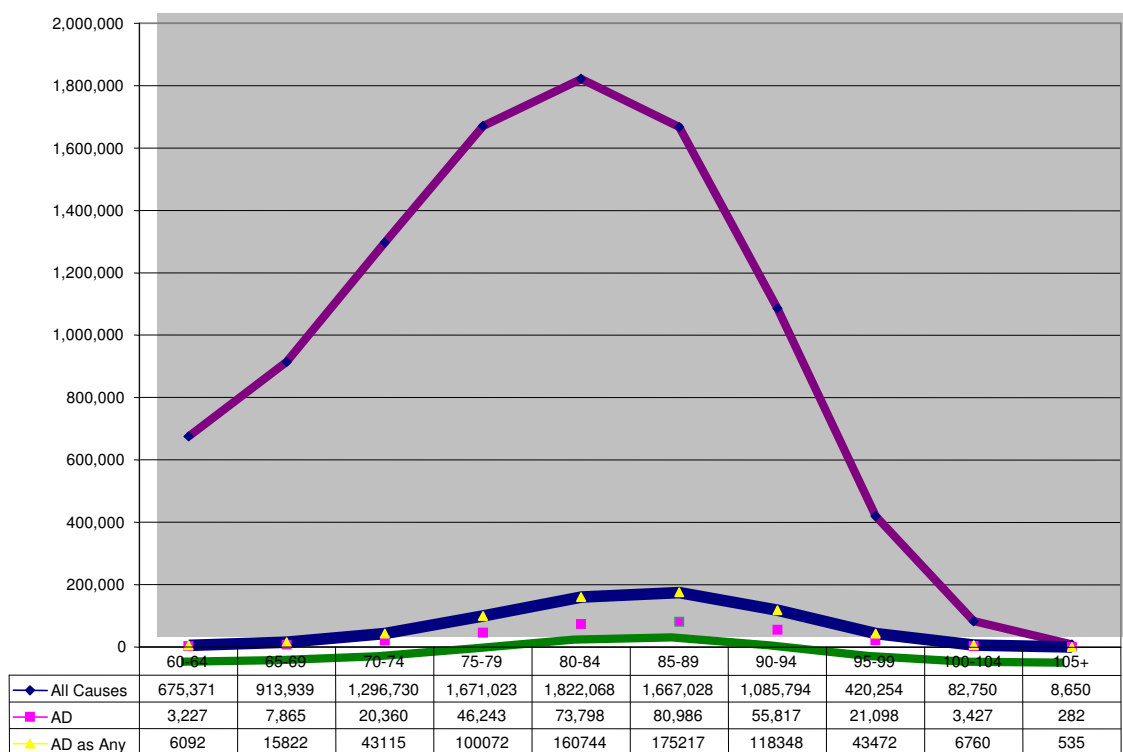
Age	Inpatient	OutPat	DOA	Nursing		Total
				Home	Residence	
60-64	240,338	56,955	9,776	41,936	163,497	675,371
65-69	332,573	66,830	10,860	73,017	209,732	913,939
70-74	471,254	80,118	12,797	143,966	277,780	1,296,730
75-79	592,291	88,406	14,041	264,688	317,474	1,671,023
80-84	600,883	80,559	12,666	408,757	299,001	1,822,068
85-89	491,482	59,554	9,806	493,759	229,530	1,667,028
90-94	270,059	30,205	5,777	402,955	130,835	1,085,794
95-99	84,919	9,103	1,988	182,582	46,834	420,254
100-104	12,891	1,379	385	40,346	9,608	82,750
105-109	1,119	119	29	4,042	1,012	8,158
110-114	72	10	4	196	66	444
115-119	10	3	0	7	9	41
120+	2	1	0	1	2	7
	2,857,555	416,287	68,353	2,014,316	1,521,883	8,968,236
	31.9%	4.6%	0.8%	22.5%	17.0%	

(Total includes unknown)

Table G.1.L Frequency of Decedents Over Age 60 by Place of Death, AD and AD as Any Condition,  
United States 1998-2002

Age	AD						AD as Any Condition					
				Nursing						Nursing		
	Inpatient	Outpatient	DOA	Home	Resident	Total	Inpatient	Outpatient	DOA	Home	Resident	Total
60-64	613	73	27	1,148	753	3,227	993	153	39	1,496	944	4,619
65-69	1,421	193	60	2,866	1,676	7,865	2,407	353	91	3,886	2,211	11,736
70-74	3,267	414	105	8,193	3,750	20,360	5,951	809	161	11,527	5,165	31,673
75-79	6,678	790	245	20,524	7,260	46,243	12,492	1,582	384	29,333	10,123	72,967
80-84	9,106	1,045	309	35,814	10,263	73,798	17,593	2,170	524	51,659	14,310	116,444
85-89	8,476	937	350	42,748	9,994	80,986	16,572	1,881	521	61,695	13,924	127,113
90-94	4,566	542	215	31,989	5,995	55,817	8,829	1,012	336	45,625	8,139	85,537
95-99	1,331	133	94	13,061	2,003	21,098	2,523	253	133	18,184	2,634	31,301
100-104	160	20	14	2,232	323	3,427	290	35	20	3,071	408	4,923
105-109	5	5	1	186	29	274	13	6	1	259	37	391
110-114	0	0	0	6	0	7	3	0	0	6	0	11
115+	0	0	0	1	0	1	2	0	0	1	0	3
Total	35,623	4,152	1,420	158,768	42,046	313,103	67,668	8,254	2,210	226,742	57,895	486,718

**Figure G.1. Frequency of Mortality by Age: All Causes, AD and AD as Any Condition, Decedents Over Age 60, United States 1998-2002**



**Figure G.2. Frequency of Mortality by Ethnicity in Decedents Over Aged 60 in the United States 1998-2002**

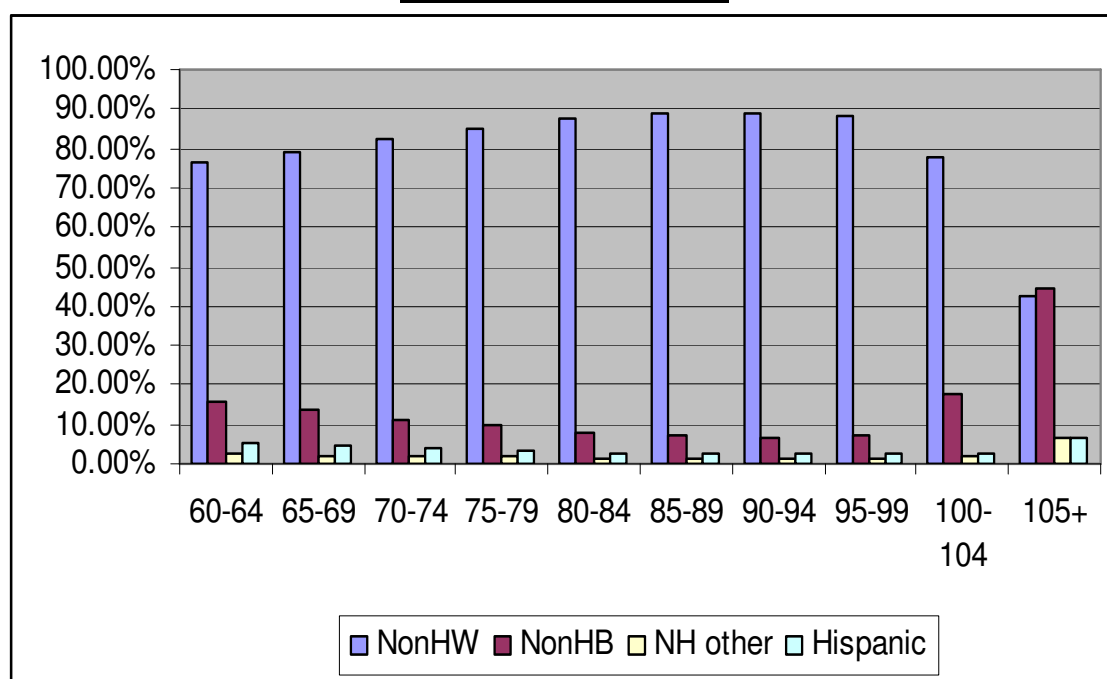


Table G.2.A: Coefficients and Percent Change in Odds Ratio  
AD as the Underlying Condition in Decedents Aged 60-64,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.272 *	-0.282 *	-0.302 *	-0.291	-0.231 *
	(-23.8)	(-24.5)	(-26.1)	(-25.2)	(-20.7)
Hispanic		-0.385 *	-0.377 *	-0.330	-0.204 **
		(-32)	(-31.4)	(-28.1)	(-18.5)
NonHispanicBlack		-0.987 *	-0.955 *	-0.924	-0.885 *
		(-62.7)	(-61.5) *	(-60.3)	(-58.7)
Married			0.273 *	0.274 *	0.132 *
			(31.4)	(31.5)	(14.1)
Widowed			0.059	0.105	-0.245 *
			(6.1)	(11)	(-21.7)
Divorced			-0.012	0.002	-0.451 *
			(-1.2)	(0.2)	(-36.3)
High School				0.021	0.104
				(2.1)	(10.9)
College				0.367 *	0.467 *
				(44.3)	(59.5)
Nonmet				-0.061	-0.035
				(-5.9)	(-3.4)
Inpatient					-0.195 *
					(-17.7)
NursingHome					2.289 *
					(886.9)
Residence					0.364 *
					(43.8)
Number	675371				
Pseudo R <sup>2</sup>	0.0014	0.008	0.0094	0.0112	0.0766
BIC:	-9025000	-9025000	-9025000	-8312000	-8314000
BIC'	-45.317	-287.303	-303.801	-306.5	-2766.751
	2721.43 0.00	2479.45 0.00	2462.95 0.00	2460.25	

\*=P<.01 \*\*=P<.05

Table G.2.B: Coefficients and Percent Change in Odds Ratio  
AD as the Underlying Condition in Decedents Aged 65-69,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.152 *	-0.164 *	-0.216 *	-0.225 *	-0.178 *
	(-14.1)	(-15.1)	(-19.4)	(-20.2)	(-16.3)
Hispanic		-0.226 *	-0.218 *	-0.117 **	0.002
		(-20.2)	(-19.6)	(-11.1)	(0.2)
NonHispanicBlack		-0.849 *	-0.811 *	-0.760 *	-0.710 *
		(-57.2)	(-55.6)	(-53.3)	(-50.8)
Married			0.283 *	0.274 *	0.173 *
			(32.8)	(31.5)	(18.9)
Widowed			-0.038	-0.017	-0.320 *
			(-3.7)	(-1.7)	(-27.4)
Divorced			0.008	0.014	-0.384 *
			(0.8)	(1.4)	(-31.9)
High school				0.073	0.114 *
				(7.5)	(12.1)
College				0.450 *	0.508 *
				(56.8)	(66.2)
Nonmet				-0.082 *	-0.066
				(-7.9)	(-6.4)
Inpatient					-0.405
					(-33.3)
NursingHome					1.943 *
					(598.2)
Residence					0.194 *
					(21.4)
Number	913939				
Pseudo R <sup>2</sup>	0.0005	0.0054	0.0072	0.0099	0.0732
BIC:	-12450000	-12450000	-12450000	-11500000	-11510000
BIC'	-30.925	-446.047	-566.455	-713.316	-6031.801
	6000.876	5585.754	5465.346	5318.485	

\*=P<.01 \*\*=P<.05

Table G.2.C: Coefficients and Percent Change in Odds Ratio  
AD as the Underlying Condition in Decedents Aged 70-74,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.149 *	-0.158 *	-0.225 *	-0.227 *	-0.167 *
	(-13.8)	(-14.6)	(-20.2)	(-20.3)	(-15.4)
Hispanic		-0.310 *	-0.304 *	-0.194 *	-0.046
		(-26.7)	(-26.2)	(-17.7)	(-4.5)
NonHispanicBlack		-0.713 *	-0.676 *	-0.630 *	-0.530 *
		(-51)	(-49.2)	(-46.7)	(-41.1)
Married			0.253 *	0.229 *	0.299 *
			(28.8)	(25.7)	(34.9)
Widowed			-0.042	-0.031	-0.159 *
			(-4.1)	(-3.1)	(-14.70)
Divorced			-0.124 *	-0.141	-0.357 *
			(-11.6)	(-13.1)	(-30)
High school				0.158 *	0.176 *
				(17.1)	(19.3)
College				0.480 *	0.503 *
				(61.6)	(65.3)
Nonmet				-0.024	-0.018
				(-2.4)	(-1.8)
Inpatient					-0.771 *
					(-53.7)
NursingHome					1.456 *
					(329)
Residence					-1.104 *
					(-66.9)
Number	1296730				
Pseudo R <sup>2</sup>	0.0005	0.0042	0.0061	0.0087	0.0754
BIC:	-1.8E+07	-1.8E+07	-1.8E+07	-1.7E+07	-1.67271
BIC'	-96.151	-832.497	-1200.19	-1579.6	-14629.1
	14532.9	13796.6	13428.88	13049.46	
*=P<.01    **=P<.05					

Table G.2.D: Coefficients and Percent Change in Odds Ratio  
AD as the Underlying Condition in Decedents Aged 75-79,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.161 *	-0.167 *	-0.251 *	-0.255 *	-0.190 *
	(-14.9)	(-15.4)	(-22.2)	(-22.5)	(-17.3)
Hispanic		-0.286 *	-0.279 *	-0.161 *	-0.001
		(-24.9)	(-24.4)	(-14.9)	(-0.1)
NonHB		-0.623 *	-0.588 *	-0.536 *	-0.405 *
		(-46.3)	(-44.4)	(-41.5)	(-33.3)
Married			0.253 *	0.225 *	0.263 *
			(28.8)	(25.2)	(30.1)
Widowed			-0.035 *	-0.032 **	-0.153 *
			(-3.4)	(-3.2)	(-14.2)
Divorced			-0.139 *	-0.147 *	-0.336 *
			(-12.9)	(-13.7)	(-28.5)
High s.				0.158 *	0.161 *
				(17.1)	(17.5)
College				0.452 *	0.455 *
				(57.2)	(57.6)
Nonmet				-0.012	-0.013
				(-1.2)	(-1.3)
Inpatient					-0.852 *
					(-57.3)
NursingH					1.187 *
					(227.6)
Resid					-1.111 *
					(-67.1)
Number	16710				
Pseudo R2	0.0007	0.0035	0.0055	0.0079	0.0708
BIC:	-2.4E+07	-2.4E+07	-2.4E+07	-2.2E+07	-2.186
BIC'	-276.404	-1431.74	-2226.24	-2989.9	-27893.9
	27617.5	26462.1	25667.63	24903.97	
*=P<.01    **=P<.05					



Table G.2.E: Coefficients and Percent Change in Odds Ratio  
AD as the Underlying Condition in Decedents Aged 80-84,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.188 *	-0.193 *	-0.283 *	-0.283 *	-0.219 *
	(-17.1)	(-17.6)	(-24.6)	(-24.6)	(-19.6)
Hispanic		-0.330 *	-0.325 *	-0.240 *	-0.067 *
		(-28.1)	(-27.7)	(-21.3)	(-6.5)
NonHispanicBlack		-0.595 *	-0.570 *	-0.516 *	-0.384 *
		(-44.8)	(-43.4)	(-40.3)	(-31.9)
Married			0.239 *	0.215 *	0.182 *
			(27)	(24)	(19.9)
Widowed			-0.022 *	-0.020	-0.172 *
			(-2.2)	(-1.9)	(-15.8)
Divorced			-0.109 *	-0.123 *	-0.317 *
			(-10.3)	(-11.5)	(-27.2)
High school				0.119 *	0.123 *
				(12.7)	(13.1)
College				0.382 *	0.387 *
				(46.6)	(47.2)
Nonmet				-0.001	-0.009
				(-0.1)	(-0.9)
Inpatient					-0.877 *
					(-58.4)
NursingHome					0.969 *
					(163.6)
Residence					-0.057 *
					(-5.6)
Number	1700343				
Pseudo R <sup>2</sup>	0.001	0.0034	0.0049	0.0068	0.0576
BIC:	-25650000	-25650000	-25650000	-23820000	-23850000
BIC'	-600.038	-2071.701	-2959.636	-3840.446	-33267.043
	32667.005	31195.342	30307.407	29426.597	

\*=P<.01    \*\*=P<.05

Table G.2.F: Coefficients and Percent Change in Odds Ratio  
AD as the Underlying Condition in Decedents Aged 85-89,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.219 *	-0.222 *	-0.305 *	-0.301 *	-0.234 *
	(-19.7)	(-19.9)	(-26.3)	(-26)	(-20.9)
Hispanic		-0.285 *	-0.282 *	-0.223 *	-0.045
		(-24.8)	(-24.6)	(-20)	(-4.4)
NonHispanicBlack		-0.488 *	-0.474 *	-0.419 *	-0.276 *
		(-38.6)	(-37.8)	(-34.2)	(-24.1)
Married			0.273	0.258 *	0.243 *
			(31.4)	(29.5)	(27.5)
Widowed			0.042 *	0.049 *	-0.061 *
			(4.3)	(5.1)	(-5.9)
Divorced			0.010	0.006	-0.133 *
			(1)	(0.6)	(-12.4)
High School				0.120 *	0.116 *
				(12.7)	(12.3)
College				0.306 *	0.300 *
				(35.8)	(35)
Nonmet				-0.013	-0.022 **
				(-1.3)	(-2.2)
Inpatient					-0.974 *
					(-62.2)
NursingHome					0.715 *
					(104.3)
Residence					-0.033 **
					(-3.3)
Number	1667028				
Pseudo R <sup>2</sup>	0.0013	0.0029	0.004	0.0052	0.0458
BIC:	-23240000	-23240000	-23240000	-21520000	-21540000
BIC'	-832.337	-1865.73	-2515.851	-3042.108	-27559.513
	26727.176	25693.783	25043.662	24517.405	

\*=P<.01    \*\*=P<.05

Table G.2.G: AD as the Underlying Condition in Decedents Aged 90-94,  
Coefficients and Percent Change in Odds Ratio

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Age	0.559 *					
	(74.8)					
Male		-0.286 *	-0.285 *	-0.349 *	-0.348 *	-0.281 *
		(-24.9)	(-24.8)	(-29.4)	(-29.4)	(-24.5)
Hispanic			-0.294 *	-0.295 *	-0.246 *	-0.068 **
			(-25.5)	(-25.5)	(-21.8)	(-6.5)
NonHispanic Black			-0.483 *	-0.479 *	-0.429 *	-0.268 *
			(-38.3)	(-38)	(-34.9)	(-23.5)
Married				0.243 *	0.238 *	0.227 *
				(27.5)	(26.8)	(25.5)
Widowed				0.038 *	0.041 *	-0.057 *
				(3.9)	(4.2)	(-5.6)
Divorced				0.111 *	0.117 *	-0.005
				(11.8)	(12.4)	(-0.5)
High school					0.108 *	0.102 *
					(11.4)	(10.8)
College					0.238 *	0.231 *
					(26.8)	(26)
Non metropolitan					-0.011	-0.018
					(-1.1)	(-1.8)
Inpatient						-1.040 *
						(-64.7)
Nursing Home						0.556 *
						(74.3)
Residence						-0.029
						(-2.8)
Number	9643607	1085794				
Pseudo R2	0.0044	0.0019	0.0035	0.004	0.0048	0.0365
BIC:	-1.5E+08	-1.5E+07	-1.5E+07	-1.5E+07	-1.4E+07	-1.4E+07
BIC'	-12172.1	-822.974	-1483.08	-1675.35	-1841.42	-14754.9
*=P<.01		**=P<.05				

Table G.2.H: Coefficients and Percent Change in Odds Ratio  
AD as the Underlying Condition in Decedents Aged 95-99,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.413 *	-0.408 *	-0.451 *	-0.450 *	-0.374 *
	(-33.8)	(-33.5)	(-36.3)	(-36.2)	(-31.2)
Hispanic		-0.282 *	-0.282 *	-0.201 *	-0.006
		(-24.5)	(-24.6)	(-18.2)	(-0.6)
NonHispanicBlack		-0.349 *	-0.348 *	-0.310 *	-0.129 *
		(-29.4)	(-29.4)	(-26.6)	(-12.1)
Married			0.280 *	0.279 *	0.245 *
			(32.3)	(32.2)	(27.8)
Widowed			0.097 *	0.097 *	-0.020
			(10.1)	(10.2)	(-1.9)
Divorced			0.170 *	0.148 *	0.009
			(18.5)	(16)	(0.9)
High school				0.121 *	0.119 *
				(12.8)	(12.6)
College				0.197 *	0.189 *
				(21.8)	(20.8)
Nonmet				-0.060 *	-0.067 *
				(-5.8)	(-6.5)
Inpatient					-1.053 *
					(-65.1)
NursingHome					0.491 *
					(63.4)
Residence					-0.049
					(-4.8)
Number	420254				
Pseudo R <sup>2</sup>	0.003	0.004	0.0045	0.0052	0.0302
BIC:	-5275000	-5275000	-5275000	-4826000	-4829000
BIC'	-488.62	-635.97	-671.608	-681.288	-4520.926
	4032.306	3884.956	3849.318	3839.638	
*=P<.01    **=P<.05					

Table G.2.I: Coefficients and Percent Change in Odds Ratio  
AD as the Underlying Condition in Decedents Aged 100-104,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.706 *	-0.691 *	-0.688 *	-0.782 *	-0.668 **
	(-50.6)	(-49.9)	(-49.7)	(-54.2)	(-48.7)
Hispanic		-0.093	-0.092	-0.114	0.112
		(-8.9)	(-8.8)	(-10.8)	(11.9)
NonHispanicBlack		-0.229	-0.229	-0.249	-0.055
		(-20.5)	(-20.5)	(-22)	(-5.4)
Married			0.138	0.465	0.599
			(14.8)	(59.3)	(82.1)
Widowed			0.201	0.360 **	0.313
			(22.3)	(43.3)	(36.8)
Divorced			0.343	0.332	0.277
			(41)	(39.4)	(32)
High school				-0.322 **	-0.325 **
				(-27.5)	(-27.7)
College				0.086	0.069
				(9)	(7.2)
Nonmet				-0.386 **	-0.395 **
				(-32)	(-32.7)
Inpatient					-1.873 *
					(-84.6)
NursingHome					0.325
					(38.4)
Residence					-0.190
					(-17.3)
Number	8158				
Pseudo R <sup>2</sup>	0.0042	0.0049	0.0058	0.013	0.036
BIC:	-71070.7	-71054.5	-71029.6	-62486.5	-62509.1
BIC'	-1.022	15.189	40.08	52.11	29.573
	-30.595	-14.384	10.507	22.537	
*=P<.01    **=P<.05					

Table G.3.A: Coefficients and Percent Change in Odds Ratio  
AD as Any Condition in Decedents Aged 60-64,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.212 *	-0.219 *	-0.238 *	-0.228 *	-0.181 *
	(-19.1)	(-19.7)	(-21.2)	(-20.4)	(-16.5)
Hispanic		-0.292 *	-0.292 *	-0.260 *	-0.154 **
		(-25.3)	(-25.3)	(-22.9)	(-14.2)
NonHispanicBlack		-0.673 *	-0.658 *	-0.636 *	-0.611 *
		(-49)	(-48.2)	(-47.1)	(-45.7)
Married			0.070 **	0.076 **	0.001
			(7.2)	(7.9)	(0.1)
Widowed			-0.100	-0.057	-0.321 *
			(-9.5)	(-5.5)	(-27.5)
Divorced			-0.127 **	-0.119 **	-0.477 *
			(-11.9)	(-11.2)	(-37.9)
High school				-0.068	0.009
				(-6.6)	(0.9)
College				0.007 *	0.298 *
				(22.3)	(34.8)
Nonmetro				0.055	-0.046
				(6.7)	(-4.5)
Inpatient					-0.168 *
					(-15.4)
NursingHome					2.081 *
					(701.6)
Residence					0.144 *
					(15.5)
Number	675371				
Pseudo R <sup>2</sup>	0.0009	0.0047	0.0052	0.0062	0.0625
BIC:	-9010000	-9010000	-9010000	-8298000	-8301000
BIC'	-37.35	-219.577	-205.213	-198.209	-3063.387
	3026.037	2843.81	-205.213	2865.178	

\*=P<.01    \*\*=P<.05

Table G.3.B: Coefficients and Percent Change in Odds Ratio  
AD as Any Condition in Decedents Aged 65-69,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.077 *	-0.086 *	-0.125 *	-0.136 *	-0.097 *
	(-7.4)	(-8.2)	(-11.7)	(-12.7)	(-9.2)
Hispanic		-0.188 *	-0.187 *	-0.120 *	-0.019 *
		(-17.2)	(-17.1)	(-11.3)	(-1.8)
NonHBlack		-0.598 *	-0.577 *	-0.542 *	-0.505 *
		(-45)	(-43.8)	(-41.8)	(-39.6)
Married			0.077 *	0.075 *	0.027
			-8.000	(7.7)	(2.7)
Widowed			-0.150 *	-0.129 *	-0.362 *
			(-13.9)	(-12.1)	(-30.4)
Divorced			-0.154 *	-0.151 *	-0.472
			(-14.3)	(-14)	(-37.6)
High school				0.004	0.044 *
				(0.4)	(4.5)
College				0.334 *	0.393 *
				(39.7)	(48.1)
Nonmetro				-0.053 **	-0.038
				(-5.1)	(-3.7)
Inpatient					-0.340 *
					(-28.8)
NursingH					1.786 *
					(-496.7)
Residence					0.026
					(-2.6)
Number	913939				
Pseudo R2	0.0001	0.0031	0.0039	0.006	0.0614
BIC:	-1E+07	-1E+07	-1E+07	-1E+07	-1E+07
BIC'	-3.248	-348.27	-403.44	-575.77	-7028.8
	7025.55	6680.53	6625.35	6453.02	

\*=P<.01 \*\*=P<.05

Table G.3.C: Coefficients and Percent Change in Odds Ratio  
AD as Any Condition in Decedents Aged 70-74,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.088 *	-0.096 *	-0.146 *	-0.145 *	-0.103 *
	(-8.5)	(-9.2)	(-13.5)	(-13.5)	(-9.8)
Hispanic		-0.260 *	-0.258 *	-0.170 *	-0.050
		(-22.9)	(-22.7)	(-15.7)	(-4.8)
NonHispanicBlack		-0.530 *	-0.509 *	-0.473 *	-0.415 *
		(-41.2)	(-39.9)	(-37.7)	(-34)
Married			0.039 *	0.019	0.003
			(3.9)	(1.9)	(0.3)
Widowed			-0.176 *	-0.164 *	-0.350 *
			(-16.1)	(-15.1)	(-29.6)
Divorced			-0.260 *	-0.276 *	-0.543 *
			(-22.9)	(-24.1)	(-41.9)
High school				0.081 *	0.106 *
				(8.4)	(11.2)
College				0.363 *	0.399 *
				(43.8)	(49)
Nonmetro				-0.003	0.001
				(-0.3)	(0.1)
Inpatient					-0.492 *
					(-38.9)
NursingHome					1.479 *
					(338.9)
Residence					-0.111 *
					(-10.5)
Number	1296730				
Pseudo R <sup>2</sup>	0.0002	0.0026	0.0037	0.0055	0.0609
BIC:	-17950000	-17950000	-17960000	-16630000	-1.7E+07
BIC':	-46.015	-744.252	-1020.892	-1421.094	-16818.8
	16772.758	16074.521	15797.881	-17218.98	

\*=P<.01 \*\*=P<.05



Table G.3.D: Coefficients and Percent Change in Odds Ratio  
AD as Any Condition in Decedents Aged 75-79,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.104 *	-0.109 *	-0.174 *	-0.177 *	-0.124 *
	(-9.9)	(-10.3)	(-16)	(-16.2)	(-11.7)
Hispanic		-0.236 *	-0.233 *	-0.141 *	-0.008
		(-21)	(-20.8)	(-13.1)	(-0.8)
NonHispanicBlack		-0.491 *	-0.469 *	-0.422 *	-0.337 *
		(-38.8)	(-37.4)	(-34.4)	(-28.6)
Married			0.033 *	0.009	0.010
			(3.4)	(0.9)	(1)
Widowed			-0.196 *	-0.194 *	-0.332
			(-17.8)	(-17.7)	(-28.3) *
Divorced			-0.280 *	-0.284 *	-0.486 *
			(-24.4)	(-24.7)	(-38.5)
High school				0.098 *	0.106 *
				(10.3)	(11.2)
College				0.335 *	0.349 *
				(39.8)	(41.7)
Nonmetro				0.013	0.010
				(1.3)	(1)
Inpatient					-0.619 *
					(-46.1)
NursingHome					1.168 *
					(221.7)
Residence					-0.204 *
					(-18.5)
Number	1671023				1,558,763
Pseudo R <sup>2</sup>	0.0003	0.0024	0.0037	0.0052	0.0576
BIC:	-23340000	-23350000	-23350000	-21670000	-2.170e+07
BIC':	-173.55	-1379.415 B	-2133.07	-2786.36	-32236.835
	32063.285	30857.42	30103.765	29450.475	
*=P<.01    **=P<.05					

Table G.3.E: Coefficients and Percent Change in Odds Ratio  
AD as Any Condition in Decedents Aged 80-84,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.130 *	-0.134 *	-0.201 *	-0.202 *	-0.145 *
	(-12.2)	(-12.6)	(-18.2)	(-18.3)	(-13.5)
Hispanic		-0.268 *	-0.267 *	-0.198 *	-0.047 **
		(-23.5)	(-23.4)	(-17.9)	(-4.6)
NonHBlack		-0.453 *	-0.436 *	-0.391 *	-0.278 *
		(-36.4)	(-35.4)	(-32.4)	(-24.3)
Married			0.028 *	0.007	0.028 *
			(2.9)	(0.7)	(2.8)
Widowed			-0.176 *	-0.174 *	-0.265 *
			(-16.1)	(-15.9)	(-23.3)
Divorced			-0.262 *	-0.276 *	-0.408 *
			(-23)	(-24.1)	(-33.5)
High s.				0.080 *	0.084 *
				(8.4)	(8.7)
College				0.304 *	0.311 *
				(35.5)	(36.5)
Nonmetro				0.035 *	0.028 *
				(3.5)	(2.8)
Inpatient					-0.726 *
					(-51.6)
NursingH					0.859 *
					(136.1)
Residence					-0.225 *
					(-20.2)
Number	1822068				
Pseudo R2	0.0005	0.0022	0.0034	0.0048	0.0487
BIC:	-3E+07	-3E+07	-3E+07	-2.36E+07	-2E+07
BIC'	-442.26	-1885.2	-2828.8	-3751.06	-39352
	38909.5	37466.6	36523	35600.717	
*=P<.01    **=P<.05					

Table G.3.F: Coefficients and Percent Change in Odds Ratio  
AD as Any Condition in Decedents Aged 85-89,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.149 *	-0.152 *	-0.215 *	-0.212 *	-0.154
	(-13.9)	(-14.1)	(-19.4)	(-19.1)	(-14.3) *
Hispanic		-0.215 *	-0.216 *	-0.169 *	-0.012
		(-19.4)	(-19.5)	(-15.5)	(-1.2)
NonHispanicBlack		-0.391 *	-0.382 *	-0.341 *	-0.219
		(-32.4)	(-31.7)	(-28.9)	(-19.7) *
Married			0.052 *	0.041 *	0.074
			(5.4)	(4.2)	(7.6) *
Widowed			-0.139 *	-0.134 *	-0.192
			(-13)	(-12.5)	(-17.5) *
Divorced			-0.183 *	-0.190 *	-0.275
			(-16.7)	(-17.3)	(-24.1) *
High school				0.075 *	0.072
				(7.8)	(7.5) *
College				0.235 *	0.233
				(26.5)	(26.3) *
Nonmetro				0.026 *	0.018
				(2.7)	(1.8) **
Inpatient					-0.798
					(-55) *
NursingHome					0.619
					(85.7) *
Residence					-0.187
					(-17) *
Number	1667028				
Pseudo R <sup>2</sup>	0.0007	0.0019	0.0027	0.0035	0.0376
BIC:	-22980000	-22990000	22990000	-21280000	-2.1E+07
BIC':	-592.737	-1647.531	-2341.851	-2826.53	-31403.3
	30810.601	29755.807	29061.487	28576.808	

\*=P<.01 \*\*=P<.05

Table G.3.G: Coefficients and Percent Change in Odds Ratio  
AD as Any Condition in Decedents Aged 90-94,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.212 *	-0.212 *	-0.261 *	-0.260 *	-0.203 *
	(-19.1)	(-9.2)	(-22.9)	(-22.9)	(-18.3)
Hispanic		-0.227 *	-0.230 *	-0.184 *	-0.025
		(-3.8)	(-20.5)	(-16.8)	(-2.5)
NonHispanicBlack		-0.374 *	-0.369 *	-0.322 *	-0.183 *
		(-8.9)	(-30.9)	(-27.5)	(-16.7)
Married			0.039 **	0.036 *	0.086 *
			(3.9)	(3.6)	(9)
Widowed			-0.138 *	-0.136 *	-0.171 *
			(-12.9)	(-12.7)	(-15.7)
Divorced			-0.077 *	-0.072	-0.130 *
			(-7.4)	(-6.9)	(-12.2)
High school				0.074 *	0.070 *
				(7.7)	(7.2)
College				0.172 *	0.170 *
				(18.7)	(18.5)
Nonmetro				0.036 *	0.030 *
				(3.7)	(3.1)
Inpatient					-0.866 *
					(-58)
NursingHome					0.451 *
					(56.9)
Residence					-0.202 *
					(-18.3)
Number	1085794				
Pseudo R <sup>2</sup>	0.0012	0.0023	0.0029	0.0033	0.0297
BIC:	-14490000	-14490000	-14490000	-13360000	-13370000
BIC':	-688.916	-1313.3622	-1653.587	-1726.322	-16358.524
	15669.608	15045.162	14704.937	14632.202	

\*=P<.01 \*\*=P<.05

Table G.3.H: Coefficients and Percent Change in Odds Ratio  
AD as Any Condition in Decedents Aged 95-99,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Age					
Male	-0.351 *	-0.348 *	-0.382 *	-0.379 *	-0.315 *
	(-29.6)	(-29.4)	(-31.7)	(-31.5)	(-27)
Hispanic		-0.184 *	-0.186 *	-0.121 *	0.054
		(-16.8)	(-17)	(-11.4)	(5.5)
NonHispanicBlack		-0.275 *	-0.274 *	-0.235 *	-0.080 *
		(-24)	(-24)	(-20.9)	(-7.6)
Married			0.084 *	0.084 *	0.125 *
			(8.7)	(8.7)	(13.4)
Widowed			-0.083 *	-0.086 *	-0.126 *
			(-7.9)	(-8.3)	(-11.9)
Divorced			0.000	-0.016	-0.077 *
			0.000	(-1.6)	(-7.4)
High school				0.085 *	0.085 *
				(8.9)	(8.9)
College				0.137 *	0.135 *
				(14.6)	(14.4)
Nonmetro				-0.016	-0.023
				(-1.6)	(-2.3)
Inpatient					-0.892 *
					(-59)
NursingH					0.371 *
					(44.9)
Residence					-0.237 *
					(-21.1)
Number	420254				
Pseudo R2	0.0024	0.0031	0.0034	0.0037	0.0244
BIC:	-5219000	-5220000	-5220000	-4775000	-4779000
BIC':	-528.58 0	-654.776	-679.195	-639.997	-4863.624
*=P<.01    **=P<.05					

Table 3.I: Coefficients and Percent Change in Odds Ratio  
AD as Any Condition in Decedents Aged 100-104,  
United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.619 *	-0.612 *	-0.607 *	-0.696 *	-0.587 *
	(-46.1)	(-45.8)	(-45.5)	(-50.2)	(-44.4)
Hispanic		-0.023	-0.022	0.005	0.231
		(-2.3)	(-2.1)	(0.5)	(26)
NonHispanicBlack		-0.099	-0.099	-0.227	-0.041
		(-9.5)	(-9.4)	(-20.3)	(-4)
Married			0.045	0.378	0.502
			(4.6)	(46)	(65.2)
Widowed			0.122	0.242	0.195
			(12.9)	(27.4)	(21.5)
Divorced			0.223	0.251	0.188
			(25)	(28.5)	(20.7)
High school				-0.406 *	-0.410 *
				(-33.3)	(-33.6)
College				0.052	0.042
				(5.3)	(4.3)
Nonmetro				-0.3388 *	-0.351 *
				(-28.7)	(-29.6)
Inpatient					-1.421 *
					(-75.8)
NursingHome					0.335
					(39.8)
Residence					-0.309
					(-26.6)
Number	8158				
Pseudo R <sup>2</sup>	0.0036	0.0038	0.0042	0.0121	0.0331
BIC:	-70331.795	-70314.28	70288.409	-61870.42	-61901.9
BIC':	-2.406	15.109	40.981	46.507	15.061
	-17.467	0.048	25.92	31.446	

\*=P<.01    \*\*=P<.05

Table 4.A: Coefficients and Percent Change in Odds Ratio  
Dying of AD Compared to Dying of Other Chronic Diseases  
in Decedents Aged 60-64, United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.262 *	-0.274 *	-0.295 *	-0.285 *	-0.228 *
	(-23.1)	(-24)	(-25.5)	(-24.8)	(-20.4)
Hispanic		-0.320 *	-0.313 *	-0.273 *	-0.169
		(-27.4)	(-26.9)	(-23.9)	(-15.6)
NonHispanicBlack		-0.980 *	-0.951 *	-0.922 *	-0.904 *
		(-62.5)	(-61.4)	(-60.2)	(-59.5)
Married			0.243 *	0.249 *	0.087
			(27.5)	(28.3)	(9.1)
Widowed			0.052	0.105	-0.283 *
			(5.4)	(11)	(-24.7)
Divorced			0.007	0.027	-0.469 *
			(0.7)	(2.7)	(-37.4)
High school				0.006	0.072
				(0.6)	(7.5)
College				0.368 *	0.452 *
				(44.4)	(57.2)
Nonmetro				-0.061	-0.032
				(-5.9)	(-3.1)
Inpatient					-0.110
					(-10.4)
NursingHome					2.312 *
					(909.4)
Residence					0.318 *
					(37.5)
Number	555193				
Pseudo R <sup>2</sup>	0.0014	0.0079	0.009	0.0111	0.0773
BIC:	-7304000	-7304000	-7304000	-6742000	-6745000
BIC'	-41.378	-274.372	-277.308	-291.292	-2704.672
	2663.294	2430.3	2427.364	2413.38	
*=P<.01    **=P<.05					

Table 4.B: Coefficients and Percent Change in Odds Ratio  
Dying of AD Compared to Dying of Other Chronic Diseases  
in Decedents Aged 65-69, United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.157 *	-0.171 *	-0.223 *	-0.233 *	-0.183 *
	(-14.5)	(-15.7)	(-20)	(-20.8)	(-16.7)
Hispanic		-0.175 *	-0.169 *	-0.073 *	0.038
		(-16.1)	(-15.5)	(-7.1)	(3.8)
NonHispanicBlack		-0.844 *	-0.808 *	-0.760 *	-0.731 *
		(-57)	(-55.4)	(-53.2)	(-51.8)
Married			0.265 *	0.260 *	0.140 *
			(30.3)	(29.7)	(15)
Widowed			-0.044	-0.018	-0.347 *
			(-4.3)	(-1.8)	(-29.3)
Divorced			0.021	0.030	-0.404 *
			(2.1)	(3.1)	(-33.2)
High school				0.063	0.104 *
				(6.5)	(10.9)
College				0.454 *	0.515 *
				(57.4)	(67.3)
Nonmetro				-0.080 *	-0.064 **
				(-7.7)	(-6.2)
Inpatient					-0.322 *
					(-27.5)
NursingHome					1.975 *
					(620.9)
Residence					0.150 *
					(16.2)
Number	770371				
Pseudo R <sup>2</sup>	0.0005	0.0055	0.0071	0.0101	0.0742
BIC:	-10350000	-10350000	-10350000	-9580000	-9585000
BIC'	-34.02	-439.021	-539.087	-704.577	-5933.865
	5899.845	5494.844	5394.778	5229.288	
*=P<.01    **=P<.05					



Table 4.C: Coefficients and Percent Change in Odds Ratio  
Dying of AD Compared to Dying of Other Chronic Diseases  
in Decedents Aged 70-74, United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.163 *	-0.174 *	-0.241 *	-0.244 *	-0.194 *
	(-15.1)	(-16)	(-21.4)	(-21.6)	(-17.6)
Hispanic		-0.275 *	-0.270 *	-0.163 *	-0.036
		(-24)	(-23.7)	(-15)	(-3.6)
NonHispanicBlack		-0.711	-0.675 *	-0.630 *	-0.577 *
		(-50.9)	(-49.1)	(-46.7)	(-43.8)
Married			0.246 *	0.225 *	0.143 *
			(27.9)	(25.2)	(15.4)
Widowed			-0.044 *	-0.030	-0.308 *
			(-4.3)	(-3)	(-26.5)
Divorced			-0.113 *	-0.128 *	-0.499 *
			(-10.7)	(-12)	(-39.3)
High School				0.151 *	0.177 *
				(16.4)	(19.4)
College				0.487 *	0.524 *
				(62.7)	(69)
Nonmetro				-0.021	-0.016
				(-2.1)	(-1.6)
Inpatient					-0.521 *
					(-40.6)
NursingHome					1.648 *
					(419.8)
Residence					-0.001
					(-0.1)
Number	1098964				1024419
Pseudo R <sup>2</sup>	0.0007	0.0043	0.0062	0.009	0.0734
BIC:	-15080000	-15080000	-15080000	-13990000	-14000000
BIC'	-118.935	-835.834	-1176.995	-1588.75	-13802.059
	13683.124	12966.225	12625.064	12213.309	

\*=P<.01    \*\*=P<.05

Table 4.D: Coefficients and Percent Change in Odds Ratio  
Dying of AD Compared to Dying of Other Chronic Diseases  
in Decedents Aged 75-79, United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.183 *	-0.190 *	-0.274 *	-0.279 *	-0.219 *
	(-16.7)	(-17.)	(-23.9)	(-24.3)	(-19.7)
Hispanic		-0.273 *	-0.266 *	-0.149 *	-0.004
		(-23.9)	(-23.4)	(-13.8)	(-0.4)
NonHispanicBlack		-0.625 *	-0.592 *	-0.541 *	-0.452 *
		(-46.5)	(-44.7)	(-41.8)	(-36.4)
Married			0.249 *	0.223 *	0.143 *
			(28.3)	(25)	(15.4)
Widowed			-0.034 *	-0.029 **	-0.268 *
			(-3.3)	(-2.9)	(-23.5)
Divorced			-0.130 *	-0.139 *	-0.456 *
			(-12.2)	(-13)	(-36)
High school				0.153 *	0.163 *
				(16.5)	(17.7)
College				0.458 *	0.475 *
				(58.1)	(60.8)
Nonmetro				-0.009	-0.013
				(-0.9)	(-1.3)
Inpatient					-0.643 *
					(-47.4)
NursingHome					1.339 *
					(281.6)
Residence					-0.088 *
					(-8.5)
Number	1402867				
Pseudo R <sup>2</sup>	0.0009	0.0038	0.0058	0.0084	0.0692
BIC:	-19450000	-19450000	-19450000	-18080000	-1.81E+07
BIC'	-359.749	-1511.165	-2263.795	-3072.496	-26236.555
	25876.806	24725.39	23972.76	23164.059	

\*=P<.01    \*\*=P<.05

Table 4.E: Coefficients and Percent Change in Odds Ratio  
Dying of AD Compared to Dying of Other Chronic Diseases  
in Decedents Aged 80-84, United States 1998-2002

	Model 5				
	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.214 *	-0.220 *	-0.308 *	-0.309 *	-0.246 *
	(-19.3)	(-19.7)	(-26.5)	(-26.6)	(-21.8)
Hispanic		-0.326 *	-0.320 *	-0.235 *	-0.070 *
		(-27.8)	(-27.4)	(-21)	(-6.8)
NonHispanicBlack		-0.599 *	-0.575 *	-0.523 *	-0.405 *
		(-45.1)	(-43.7)	(-40.7)	(-33.3)
Married			0.237 *	0.215 *	0.148 *
			(26.8)	(23.9)	(15.9)
Widowed			-0.018	-0.013	-0.208 *
			(-1.7)	(-1.3)	(-18.8)
Divorced			-0.098 *	-0.109 *	-0.354 *
			(-9.3)	(-10.4)	(-29.8)
High school				0.115 *	0.120 *
				(12.2)	(12.7)
College				0.391 *	0.401 *
				(47.9)	(49.4)
Nonmetro				0.002	-0.009
				(0.2)	(-0.9)
Inpatient					-0.760 *
					(-53.2)
NursingHome					1.025 *
					(178.6)
Residence					-0.113 *
					(-10.7)
Number	1498866				1400732
Pseudo R <sup>2</sup>	0.0013	0.0039	0.0054	0.0075	0.0586
BIC:	-20730000	-20730000	-20730000	-19280000	-19300000
BIC'	-774.382	-2252.27	-3089.407	-4033.774	-32213.735
	31439.35	29961.47	29124.33	28179.96	
*=P<.01    **=P<.05					

Table 4.F: Coefficients and Percent Change in Odds Ratio  
Dying of AD Compared to Dying of Other Chronic Diseases  
in Decedents Aged 85-89, United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.250 *	-0.253 *	-0.334 *	-0.330 *	-0.265 *
	(-22.1)	-33.057	(-28.4)	(-28.1)	(-23.3)
Hispanic		-0.295 *	-0.293 *	-0.231 *	-0.059 **
			(-25.4)	(-20.7)	(-5.7)
NonHispanicBlack		-0.499	-0.486 *	-0.431 *	-0.302 *
			(-38.5)	(-35)	(-26.1)
Married			0.269 *	0.255 *	0.201 *
			(30.9)	(29)	(22.3)
Widowed			0.047 *	0.055 *	-0.101 *
			(4.8)	(5.6)	(-9.6)
Divorced			0.028	0.024	-0.165 *
			(2.8)	(2.5)	(-15.2)
High school				0.120 *	0.118 *
				(12.8)	(12.5)
College				0.317 *	0.315 *
				(37.3)	(37.1)
Nonmetro				-0.005	-0.018 **
				(-0.5)	(-1.8)
Inpatient					-0.840 *
					(-56.8)
NursingHome					0.768 *
					(115.6)
Residence					-0.096 *
					(-9.2)
Number	1336410				1245886
Pseudo R <sup>2</sup>	0.00 18	0.00 36	0.0046	0.006	0.0456
BIC:	-18240000	-18240000	-18240000	-16920000	-16940000
BIC'	-1073.051	-2149.349	-2754.339	-3311.639	-25890.502
	24817.45	23741.15	23136.16	22578.86	
*=P<.01    **=P<.05					

Table 4.G: Coefficients and Percent Change in Odds Ratio  
Dying of AD Compared to Dying of Other Chronic Diseases  
in Decedents Aged 90-94, United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.313 *	-0.313 *	-0.374 *	-0.373 *	-0.311 *
	(-26.9)	(-26.8)	(-31.2)	(-31.1)	(-26.8)
Hispanic		-0.321 *	-0.322 *	-0.271 *	-0.098 *
		(-27.5)	(-27.6)	(-23.7)	(-9.3)
NonHispanicBlack		-0.496 *	-0.492 *	-0.443 *	-0.300 *
		(-39.1)	(-38.8)	(-35.8)	(-25.9)
Married			0.237 *	0.232 *	0.181 *
			(26.7)	(26.1)	(19.9)
Widowed			0.041 *	0.045 *	-0.101 *
			(4.2)	(4.6)	(-9.6)
Divorced			0.122 *	0.128 *	-0.045
			(13)	(13.6)	(-4.4)
High school				0.111 *	0.105 *
				(11.7)	(11)
College				0.251 *	0.248 *
				(28.6)	(28.2)
Nonmetro				0.000	-0.012
				(0)	(-1.2)
Inpatient					-0.886 *
					(-58.8)
NursingHome					0.608 *
					(83.7)
Residence					-0.093 *
					(-8.9)
Number					783533
Pseudo R <sup>2</sup>	0.0024	0.0042	0.0047	0.0057	0.0358
BIC:	-11110000	-11110000	-11110000	-10250000	-10260000
BIC'	-976.383	-1681.537	-1859.312	-2051.124	-13508.347
	12531.96	11826.81	11649.04	11457.22	
*=P<.01    **=P<.05					

Table 4.I: Coefficients and Percent Change in Odds Ratio  
Dying of AD Compared to Dying of Other Chronic Diseases  
in Decedents Aged 100-104, United States 1998-2002

	Model 1	Model 2	Model 3	Model 4	Model 5
Male	-0.747 *	-0.729 *	-0.721 *	-0.817 *	-0.702 **
	-52.600	(-51.8)	(-51.4)	(-55.8)	(-50.4)
Hispanic		-0.086	-0.090	-0.111	0.128
		(-8.3)	(-8.6)	(-10.5)	(13.6)
NonHispanicBlack		-0.272	-0.271	-0.315	-0.131
		(-23.8)	(-23.8)	(-27)	(-12.3)
Married			0.056	0.417	0.579
			(5.8)	(51.8)	(78.5)
Widowed			0.194	0.359 **	0.306
			(21.5)	(43.1)	(35.8)
Divorced			0.380	0.371	0.288
			(46.2)	(44.9)	(33.4)
High school				-0.349 **	-0.336 **
				(-29.5)	(-28.5)
College				0.078	0.078
				(8.1)	(8.1)
Nonmetro				-0.319 **	-0.350 **
				(-27.3)	(-29.6)
Inpatient					-1.725 *
					(-82.2)
NursingHome					0.341
					(40.7)
Residence					-0.275
					(-24)
Number	5158				5158
Pseudo R <sup>2</sup>	0.00 51	0.00 62	0.0072	0.01 45	0.0366
BIC:	-47903.33	-47888.51	-47864.59	-42064.09	-42081.956
BIC'	-2.559	12.263	36.184	48.287	30.422
	-32.981	-18.159	5.762	17.865	

\*=P<.01 \*\*=P<.05

Table 5.A: Coefficients and Percent Change in Odds Ratio  
AD as the Underlying Condition in Decedents  
Aged 60-105+, United States 1998-2002

65-69	-0.020	(-2)
70-74	0.589 *	(80.1)
75-79	1.168 *	(221.4)
80-84	1.562 *	(376.7)
85-89	1.752 *	(476.7)
90-94	1.812 *	(512)
95-99	1.787 *	(496.9)
100-104	1.367 *	(292.5)
105+	0.624	(86.7)

Age 60-64 used as the standard

Number of obs = 9643607

Pseudo R2 = 0.0317

BIC: -1.524e+08 BIC': -87307.777

Table 5.B: Coefficients and Percent Change in Odds Ratio  
AD as Any Condition in Decedents Aged 60-105+,  
United States 1998-2002

65-69	0.020	(2)
70-74	0.675 *	(96.4)
75-79	1.276 *	(258.2)
80-84	1.678 *	(435.6)
85-89	1.868 *	(547.5)
90-94	1.903 *	(570.8)
95-99	1.843 *	(531.3)
100-104	1.373 *	(294.9)
105+	0.832 **	(129.8)

Age 60-64 used as the standard

Number of obs = 9643607

Pseudo R2 = 0.0365

BIC: -1.514e+08 BIC': -140654.382

Table 5.C: Coefficients and Percent Change in Odds Ratio  
AD as Underlying Condition in Decedents Aged 60-105+,  
Mexican and Cuban Comparisons, United States 1998-2002

Mexican	-0.517 *	(-40.4)	176,241
Mexican in SW	-0.561 *	(-43)	142,283
Cuban	-0.024	(-2.4)	48,244
Cuban in Florida	0.012	(1.2)	37,323

Table 5.D: Coefficients and Percent Change in Odds Ratio  
AD as Underlying Condition in Decedents Aged 60-105+,  
By Ethnicity and Age, United States 1998-2002

	60-64	60-64	70-74	75-79	80-84	85-89	90-94	100-104
Mexican	-0.760 *	-0.342 *	-0.435 *	-0.351 *	-0.410 *	-0.376 *	-0.416 *	-1.385
	(-53.2)	(-29)	(-35.3)	(-29.6)	(-33.6)	(-31.3)	(-34.1)	(-75)
PuertoRican	-0.060	-0.035	-0.175	-0.342 *	-0.595 *	-0.279 *	-0.336 *	0.371
	(-5.9)	(-3.5)	(-16.1)	(-29)	(-44.8)	(-24.3)	(-28.5)	(44.9)
Cuban	0.093	0.130	-0.033	0.061	0.022	-0.214 *	-0.218 *	-0.086
	(9.8)	(13.9)	(-3.3)	(6.3)	(2.2)	(-19.2)	(-19.6)	(-8.2)
OtherHispanic	-0.181	-0.167	-0.241 *	-0.299 *	-0.317 *	-0.189 *	-0.207 *	0.701
	(-16.5)	(-15.4)	(-21.4)	(-25.9)	(-27.2)	(-17.3)	(-18.7)	(101.5)
NonHisBlack	-0.978 *	-0.841 *	-0.705 *	-0.615 *	-0.587 *	-0.481 *	-0.481 *	-0.256
	(-62.4)	(-56.9)	(-50.6)	(-45.9)	(-44.4)	(-38.2)	(-38.2)	(-22.6)



## VITA

Name: Mary Ann Davis

Address: Sociology Department  
Texas A&M University  
College, Station, Texas

Email Address: marydavis@tamu.edu

Education: Mary Ann Davis, B.A., Lamar University, 1969  
MSSW, University of Texas at Arlington, 1971  
Ph.D., Texas A&M University, 2006